THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—19TH YEAR. SYDNEY, SATURDAY, MAY 28, 1932. No. 22.

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E. H. Embley Memorial Lecture.[1]

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IT is customary, and it is fitting, that the first of a series of memorial lectures should be of a biographical character. I can lay claim at least to one qualification not always possible with biographers, and that is close personal friendship with the subject of study.

Edward Henry Embley was born in Castlemaine, Victoria, Australia, on February 27, 1861. His parents were both from Gloucestershire, England, and belonged to that army of pioneers who, impelled by the love of adventure and the lure of gold, poured into Australia and precipitated this country into a nation. At the time of young Embley's boyhood the old glories of the gold rush had faded from Castlemaine and the town was already on that downward path, fortunately often temporary, which old mining towns always experience. Times were bad and pocket money was scanty. In his early years, he was a delicate child, but full of spirit and anxious to join in games often beyond his strength, quick-eyed and rather hot-tempered. Sometime about his ninth or tenth year, a little book on practical chemistry came into his possession. This set the busy imagination going and may be said to have influenced his whole career. He had his little laboratory in an outhouse, he saved all his pennies to buy the commoner acids and alkalis, and devoted all his leisure to this great adventure, in which the apparatus had to be improvized. His youthful chemistry was not merely the usual home manufacture of hydrogen, oxygen,

[1] Delivered in the Medical Society's Hall, Melbourne, March 17, 1932.

chlorine, sulphuretted hydrogen and ammonia, punctuated by explosions, but a deliberate effort to produce pure products and active principles including starches, gums and distilled essences. Though his first love was eclipsed for a time by an orgy of miniature hydraulic engineering, he remained true to his early chemical aspirations all his boyhood and youth. What may be called his first great sorrow was the loss of his little laboratory and all his preparations through a storm of wind which wrecked the outhouse and its contents beyond hope of repair.

His early schooling was at Castlemaine and was not as congenial as it might have been. Embley was left-handed and his teacher systematically and forcibly tried to correct this, but without success. To this attempted correction of what apparently was uncorrectable Embley in after life attributed, whether rightly or wrongly let neurologists decide, his lack of fluence in speech and a dread of public utterance which he never conquered. A new headmaster of a more sympathetic type brought some stimulus to bear, and great was the boy's triumph when in a school of some three hundred pupils he sent in the only correct solution of an arithmetical problem and won the coveted prize of two shillings and sixpence.

Between his thirteenth and fourteenth year mysterious endocrine influences changed his physique and even to some degree his character. He rapidly acquired strength and confidence and soon began to find delight in long tramps or fishing or shooting excursions with occasional camping in the bush accompanied by a congenial companion. His elder brother who had many similar intellectual gifts was separated by a sufficient number of years to prevent that intimate companionship which a boy looks for and enjoys. Also the irritable temper gave place to the composure which was so characteristic of his maturer years. About the age of fourteen he was apprenticed to a Castlemaine pharmacist who soon afterwards failed in business. Young Embley then went to Bendigo and served his time in a chemist's shop and was able to continue his education at the Bendigo High School. He passed the qualifying examination in pharmacy in Melbourne on December 4, 1879, but was not formally admitted to the profession until March 8, 1882, when he was twenty-one years old. The only employment that presented itself was to take charge, with the offer of purchase on terms, of a small chemist's business in Melbourne which had been neglected. He succeeded in rehabilitating this concern and gained sufficient income to marry on December 27, 1883, his choice being Miss Lydia Cox, born in Bath, England, who made an ideal helpmeet both in his early struggles and in his subsequent successes. His ambitions were now directed towards medicine, so he matriculated in the University of Melbourne in 1884 and managed to attend classes and keep the business going until he gained the qualifying degrees of M.B., B.S. in 1889, obtaining them in what was then the minimum time for the Melbourne Medical School. To do this meant hard work, but the happiness of his home life made it possible. Very soon after qualifying he started practice in Latrobe Street, Melbourne, which remained his professional headquarters until he retired in 1920. His practice was what is usually designated general, but soon anæsthesia began to attract his special interest, and his proficiency in this led to a large anæsthetic practice. A pleasant association with the surgeon, Mr. R. A. Stirling, remained unbroken until the death of the latter. He was soon appointed Honorary Anæsthetist to the Melbourne Hospital, a position which he held until 1917, when he resigned and was made Consulting Anæsthetist, an honour he retained till the end. Embley's experimental researches have so outshone his other activities that there is a danger of our forgetting what occupied most of his time, the daily round of duties as medical practitioner. He was truly "the beloved physician" to his patients, bringing sympathy and begetting confidence and acting as counsellor in many family matters other than medical. The gentle, modulated voice, the unhurried assurance in his actions, the serenity of his mind, and above all the punctilious rectitude of his conduct towards patients and colleagues, compelled respect and admiration.

Embley's earlier publications were, as is usual, purely descriptive: "The Treatment of Typhoid with β Naphthol" (1894); "A Case of Aneurism of the Heart with a New Diagnostic Sign" (1895) (this was a musical note throughout the cardiac cycle due to a tag vibrating in the blood current); "A Case of Myxœdema" (1895); "A Case of Puerperal Septicæmia" (1896); "Tetanus Successfully Treated with Anti-Tetanic Serum" (1899).

In the *Intercolonial Medical Journal* of 1896, there was printed an article by E. H. Embley which gives the key-note to all his subsequent investigations. It is entitled "The Question of Safety in Syme's Teaching in Chloroform Anæsthesia". Here, with his experience of the human patient alone to guide him, he announced his conviction that the great Scotch surgeon Syme and the renowned Hyderabad Commission on Chloroform were wrong in their conclusions. He had succeeded in convincing himself, but it was another matter to convince the world. Here it is necessary that I drop for a space my rôle as biographer and tell briefly the curious story of chloroform. Humphry Davy had shown in 1798 that breathing nitrous oxide produced anæsthesia, and he suggested that surgical operations could be carried out during the stage of unconsciousness. This hint was adopted by the American dentist Wells in 1844. In 1846 the Americans, Morton and Warren, carried out an operation on the human patient under ether anæsthesia. The inferior prestige of American surgery at the time, unfortunately for humanity, led to the eclipse of ether. The French physiologist, Flourens, used chloroform for anæsthetizing lower animals, and in 1847 Simpson, of Edinburgh, brought chloroform into general use for human surgery. At first there was no idea of danger associated with the use of this agent, indeed sending

somebody to sleep with chloroform was a drawing room entertainment until a very decorous gentleman uttered most unexpectedly indecorous words as he was "going under". But soon fatalities came in the operating theatre, increasing in number until the public became thoroughly alarmed, and producing that dread of operation which has lasted until this day, when it is almost as safe to enter anæsthesia as it is to enter a railway train. Simpson had his share of these fatalities, and he arrived at the conclusion that there were two causes of death: (i) Syncope or sudden failure of circulation; (ii) asphyxia or failure of respiration. As we now know, thanks largely to Embley, Simpson showed considerable sagacity in this judgement. But, unfortunately for the profession and humanity, Syme, of Edinburgh, took another view, and his famous aphorism, "attend to the respiration, never mind the pulse", dominated the administration of chloroform throughout Scotland and part of England. In 1855 Syme published a lecture on chloroform which was accepted as gospel by his colleagues and pupils. In it occur the words, "the more rapidly chloroform is given the better . . . we are guided as to the effect, not by the circulation, but entirely by the respiration. You never see anybody here with his fingers on the pulse while chloroform is given." In London the opposite view was largely held, though by no means universally. "Watch the pulse" was the advice here given to the anæsthetist. Syme's views, however, were upheld in London by all the authority associated with the great name of Joseph Lister.

The first experimental researches and therefore the first scientific approach to the problem were made by Snow in England and Flourens in France. Flourens did good service by drawing attention to the toxicity of chloroform. Snow, with very limited appliances and resources at his disposal, reached the correct conclusion that though the respiration usually ceases before the heart, sometimes the heart is the first to stop. In 1855 a chloroform commission in Paris supported Syme's contention that respiration failed before the heart. In 1879 the British Medical Association appointed a committee in Glasgow to experiment with anæsthetics. This committee included the distinguished chemist, William Ramsay, and the physiologist, John McKendrick. They reached the conclusions that lowering of the blood pressure is due to weakening of the heart's action, that this effect is given by chloroform and not by ether, and that death may occur any time during chloroform inhalation by sudden stoppage of the heart. They were the first, I believe, to bring into consideration the action of chloroform on the heart through its nerve supply.

The report of the Glasgow committee was widely accepted by the profession. Syme's authority seemed to have undergone eclipse. But a perfervid Scot, a pupil and house surgeon of Syme's, Surgeon Lieutenant-Colonel Lawrie, thought otherwise. It was he who originated the first Hyderabad Commission with himself a director. What he lacked in physiological training, and this was zero, and also in scientific temper, he made up in aggressive assurance, plunging into experimentation with animals without realizing that this required a long discipline of training. Naturally his conclusions supported Syme. His criticisms of the Glasgow committee were sweeping: "Common sense was cast to the winds and two erroneous principles became established on a pseudo-scientific basis." "The effect of the Glasgow committee was enormous, far-reaching and disastrous." Colonel Lawrie's methods might succeed with army subordinates or with Babus, but the medical world did not accept him at his own valuation, whereupon the second Hyderabad Commission was formed through the generosity of the Nizam. A skilled experimentalist was regarded as necessary and the choice fell on Lauder Brunton. This was an unfortunate selection in view of the fact that Britain at the time possessed a galaxy of physiologists, any one of whom would have succeeded better than Brunton, who was one of those amphibious gentlemen who acquire fame in either element by their supposed preeminence in the other. The beautifully bound report of this commission was issued in 1891. The conclusions embodied in this report are certainly remarkable. Failure of respiration is the only means by which the heart's safety is jeopardized; the heart never stops before respiration; vagus action on the heart is beneficial, preventing a too great distribution of the chloroform; chloroform does not directly injure the heart substance; the fall of blood pressure during anæsthesia is beneficial to the heart; the Glasgow committee used faulty methods and ascribed to chloroform what was really due to asphyxia. Now each of these conclusions is not only wrong, but wholly wrong. How much of the blame is to be ascribed to the climate, to illness and to the Nizam's hospitality is hard to apportion, but lack of scientific training must assuredly be the chief factor.

The report of the Hyderabad Commission evoked a storm of protest from physiologists, particularly British and French. The most trenchant of these attacks came from Professor (now Sir Leonard) Hill, who turned upon Colonel Lawrie some of Colonel Lawrie's own invective and directed against Lauder Brunton's experimental work the most withering criticism. An examination of the records of the Hyderabad Commission fully justify Leonard Hill's attack. The experimentation was clumsy and amateurish. A student of physiology today who produced such faulty tracings would be discouraged from further study in the subject. Leonard Hill and his fellow physiologists, McWilliam, Gaskell, Shore, Waller, McKendrick, Richet and others may have shaken public confidence in the Hyderabad Commission, but Colonel Lawrie was a fighter and his blood was up. When observations were recorded that ran counter to his views, he ascribed such to the incompetence of the observer. Lawrie still had a following and the Syme tradition was still strong in certain quarters, especially where Lister had influence. In Melbourne

at the time deaths under chloroform were frequent and the doctrine of Syme still survived. In the very same journal in which Embley's first doubts were expressed (1896) Mr. Hamilton Russell, who brought to Melbourne the Listerian tradition in surgical technique, had an article immediately preceding Embley's in which the Syme attitude was vigorously defended and maintained. "Watch the breathing and let the pulse alone. For the pulse is the Mephistopheles of the chloroformist." Still more definite were the following two dicta: "(1) In no case can the finger on the pulse be of the slightest value; (2) it can do, and frequently does, mortal harm." In justification of Mr. Russell's attitude, it may be stated that this is true enough of anæsthesia when the patient is well under and being operated on by the surgeon, and Mr. Russell was drawing on his experience as a surgeon and not as an anæsthetist. It was amid this welter of opinion that Embley's experimental work was carried out.

A biographer is unconsciously, and almost inevitably, biased in favour of his subject.[1] Though Embley's friends would like full credit to be paid him for the success of the work on chloroform, it is necessary in the interests of truth that that credit should be shared. When I first met Embley, in 1904, he was a skilled experimentalist in a department of experimentation which is fairly difficult. I have met professors of physiology who were his inferiors. Now it was obvious to me from the start whence his training and inspiration had come. Each school has its own devices, tricks and procedures, and those of Embley were strongly reminiscent of Guy's Hospital, and the former close association of C. J. Martin and E. H. Starling were always forced upon my notice when Embley was at work. He had previously no training whatever in experimental physiology and very little knowledge of physiological theory. He had, indeed, no original ambitions in that field, for as a matter of fact he went to the University to find help in his efforts to prepare a purer ether for inhalation. Worry concerning the deaths at the Melbourne Hospital under chloroform led to his discussing this topic with Professor C. J. Martin, and soon he was induced to embark on a research which grew and grew until it spread over nearly four years and involved some 280 experiments. The technique he had to acquire slowly with many trials, delays and disasters. At one time it looked as if he never could overcome his lack of proper preparation for the task. "Embley had, however, two of the essential qualities of a discoverer, an unadulterated curiosity to know how things worked and inexhaustible patience and persistence."[2] I have myself seen many instances of that patience. When a stupid laboratory boy by a thoughtless or clumsy act undid the work of weeks, when I might have expected, and certainly should have excused, an outburst of temper, all I heard was a gentle "tut, tut" and then he would begin to build up the experiment once more. His instructor, Professor Martin, could not always be at hand, owing to teaching and administrative duties, and, further, Embley's practice kept him busy during the week days, so that he was forced to devote to the research his Sundays when the skilled mechanic (the late Mr. Grayson) was absent and Professor Martin not always able to attend. But patience and the spirit of the discoverer eventually triumphed. May I quote here from an obituary notice which I wrote in 1924 at the request of the Editor of THE MEDICAL JOURNAL OF AUSTRALIA:

> The four prime qualities of the man of science were strongly shown in him—courage, imagination, accuracy, persistence. I never saw these better displayed than in one of his few failures. He was endeavouring to find some quick reliable method of determining the concentration of chloroform in blood and had decided to use the vapour pressure. This meant, of course, fixing the oxygen and carbon dioxide which would otherwise be liberated at reduced pressures. Then out of odd bits of glass tubing, spare taps and pieces of a broken Toepler pump he built up a beautiful tensimeter in a windowed thermostat which gave the vapour pressure of liquids with accuracy. Alas, the small amounts of chloroform and the disturbance due to dissolved nitrogen rendered the method useless. . . Another episode dwells in my memory. Embley was investigating the action of anæsthesia on the peripheral circulation and was using a bowel plethysmograph. In the course of a friendly argument I advanced the criticism that he was not allowing for gravity and to prove his point he ought to have the animal prone with the plethysmograph pointing downwards. Next day I found Embley at work with animal and apparatus upside down and pointing proudly to his positive result.

A fact that must not be forgotten is that the Physiological Laboratory in Melbourne was then, owing to general financial depression, very badly equipped. Professor Martin had a genius for devising efficient apparatus out of odds and ends of machine parts and scraps of metal and glass, and Embley learned some of his art.

C. J. Martin's share in the great investigation on chloroform cannot be assessed, as the one man who could give the information prefers to maintain a modest silence. I have, however, been able to obtain from him the following characteristic statement which does credit to the writer as well as his subject:

> In the course of that work two useful methods were devised and exploited for the first time and subsequently used by others.
>
> (1) The use of the lungs to oxygenate an artificial circulation of defibrinated blood (still the only satisfactory method).
>
> (2) The heart-lung preparation subsequently used with such good effect by Starling and Anrep.
>
> For these I must claim some share and I suppose I did give him a good deal of help, but his simple child-like curiosity, determination and patience, and invariable good temper when disaster occurred, excited my admiration and endeared him to me and I should like to do it all over again.

Embley's great contribution to medical science was entitled "The Causation of Death During the Administration of Chloroform" and appeared in *The British Medical Journal* of April 5, 12 and 19, 1902. What may be said of its conclusions? Briefly

[1] An exception is furnished by the Reverend Griswold who foully traduced the good name of Edgar Poe.

[2] From a letter written to me by Sir Charles Martin.

it may be stated that it went over the whole work of the Hyderabad Commission and defeated it in detail. He showed that heart muscle is very sensitive to chloroform poisoning; that the drug raises the excitability of the vagus; that deaths in the induction stage of anæsthesia (which the Hyderabad Commission ignored) are syncopal and unconcerned with respiration; that failure of respiration is mainly due to fall of blood pressure; and that in the post-induction stages of anæsthesia there is a general depression of all activities and no longer syncope through excited vagus action. The only important point which he may be said to have missed was the fibrillation of the heart caused by deep chloroform poisoning. The publication of this research, together with the display of the tracings in London by Professor Martin, gave the *coup de grace* to the Hyderabad Commission. That arrest of the heart through vagal action was the cause of fatal syncope was not fully accepted by physiologists. Further cogitation and further experimentation, this time under my observation, though I cannot claim any great share in direction, led to a modification of his views. There was not only an arrest of the muscular activity of the heart, but an arrest of the muscular activity of the arterioles; in his own words:[1]

> Chloroform syncope is an inhibition of the heart by the vagus nerves combined with inhibition of splanchnic vaso-constriction. . . I would define syncope occurring during the administration of anæsthetic agents as sudden, complete or partial cessation of circulation due, in the majority of instances, to combined vagal inhibition of the heart and of inhibition of splanchnic vaso-constriction, and in the minority of instances to splanchnic vaso-constrictor inhibition alone.

Embley's next publication was in collaboration with Professor Martin. He had declared in his chloroform paper that this drug led to a dilatation of the arterioles through direct poisonous action. Professor (afterwards Sir Edward) Schafer now came forward with proof that chloroform added to the perfusion fluid passing through the body of a frog led to a constriction of the arterioles. Embley and Martin cleared up the discrepancy. With the aid of a most ingenious artificial circulation apparatus they showed that in the concentrations to which chloroform would attain in the blood when it was inhaled, dilatation of the arterioles would alone be produced, whilst Schafer's results were due to direct irritation of the chloroform in concentrations impossible in the course of anæsthesia by inhalation.

From chloroform Embley next turned his attention to other volatile anæsthetics. In "The Pharmacology of Ethyl Chloride", published in the *Proceedings of the Royal Society, London*, in 1906, he showed, using the same experimental methods, that this drug produced much the same effects as chloroform, but in less degree. Ethyl chloride, he declared, had one-fourth the action of chloroform on the vagus and one-nineteenth the action on the heart muscle. "It is on this difference between the action of the two anæsthetics that the relative safety of ethyl chloride rests". "The Action of Ether on the Circulation" was published in the *Biochemical Journal* in 1910. Here he pointed out, as others have done, that ether is in every way safer than chloroform. "With chloroform (cardiac) inhibition is frequent and persistent; with ether infrequent and transitory." Ether, Embley announced, had one-fiftieth of the toxic action of chloroform on the heart. (Cushny by another procedure found the relation to be one-thirty-sixth.) Ether unfortunately produces undue relaxation of the arterioles, and this led Embley to support the "closed" method of administration where a mild asphyxia keeps the arterioles constricted and therefore prevents fall of blood pressure.

In his later publications Embley collated and classified his results and expounded them in a clear manner. The various causes of death under anæsthesia he grouped under four headings: (i) syncope, (ii) excessive intoxication, (iii) shock, in which he recognized a nervous factor and an adrenal factor, but missed the histamine contribution, (iv) pathological states and various accidents, such as flooding of the bronchi. He gave the treatment which each separately requires.

Some of his researches ended, as such often will, in failure. For instance, he tried to resuscitate an over-chloroformed dog by forcibly injecting saline solution into a proximal carotid in the hope that this would reach the coronaries. But alas, the pulmonary dropsy it produced prevented its application to the human being. He made many minor discoveries which he did not think worthy of publication, but which have brought credit to other investigators. Thus he quite independently found that rise of pressure in the great veins leads to reflex acceleration of the heart. This is now called the Bainbridge reflex. I remember well his drawing my attention several times in 1909 to a curious reaction of the heart. When the coronary artery was distended with anything, saline solution, oil, or even his own breath from a pipette, the heart would often start beating if quiescent, or would be strengthened if beating feebly.

The old love of chemistry never forsook him. His last published paper was on "Formaldehyde in Ether" (1916), and he carried out a purely chemical research on the absorption of ether by strong sulphuric acid and the subsequent liberation of pure ether on adding water. Had he not been dedicated to medical and physiological work, I am convinced that he would have gained success as a technical chemist. He had a singularly sure instinct where chemical technique was concerned.

In 1920 Embley was forced by feeble health to resign from his medical practice, and the remaining four years of his life witnessed the tragedy of steadily advancing disease which he bore with unconquerable serenity of mind. He died on May 9, 1924.

Embley's happy married life was marred by one great sorrow, the death from illness of an infant

[1] *Australian Medical Journal*, January 24, 1914.

son. The subject obviously awakened such poignant feelings that I forbore to question. He was brought up in the Church of England, but his wife and two daughters, who now survive him, attended the Church of Christ. His own Sunday devotions were conducted in a temple of truth which happened to take the form of a physiological laboratory.

His merit, it is pleasing to note, did not pass unrecognized during his lifetime. A thesis on anæsthesia gained the M.D. of Melbourne University in 1901. When the David Syme Prize was instituted as a recognition of the best research work in Australia conducted during the previous two years, Embley was the first recipient. He kept the medal, but handed half the money over to me for the funds of my laboratory. The value of his chloroform work published in 1902 was recognized at once. In the words of *The British Medical Journal*, it was "a distinct advance in knowledge". A brass memorial tablet has been erected in the Medical Society's Hall in Melbourne and another in the Wilson Hall of the University, each bearing witness to his success as a scientist and to his nobility of character. In 1929 a Scroll of Recognition was handed in person to the Chancellor of the University of Melbourne by Dr. F. H. McMechan, Editor-Secretary-General of the International Anæsthesia Research Society. This posthumous honour, rarely bestowed, pays glowing tribute to Embley's life and work. When the eighth Annual Congress of Anæsthetists took place in Chicago in the same year a special Edward Henry Embley Memorial Dinner was held on the evening of Thursday, October 17. The menu card was graced with a good reproduction of his photograph. A number of subscribers, generously helped by his son-in-law, Mr. Mitchell, have founded the memorial lectures now begun, and are able to present an Embley Gold Medal to be awarded annually to a student of medicine in his sixth year as a result of a competitive examination in the subject of anæsthesia. The memorial lectures of the future will take forms which naturally I cannot foretell, but I am reasonably certain that the subject he loved will be dealt with in a manner that will benefit the profession and humanity, and doubtless this biographical sketch will be much improved on and expanded. As the years pass the number of those who knew him personally must progressively lessen, but those who have had that privilege feel that there was a sweetness of disposition and an attraction about Edward Henry Embley which no skill in words can convey or perpetuate.

I should like to express my thanks for valuable information received, and unobtainable otherwise, to Mrs. Embley and her two daughters, and to Mr. J. T. Embley, the late Dr. Embley's surviving brother.

Bibliography of E. H. Embley's Publications.

The following is a bibliography of Embley's publications. I cannot claim that it is complete. It includes all that I have been able to discover.

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(2) "A Case of Aneurism of the Heart and Probable Diagnostic Sign of Such Condition", *Australian Medical Journal* (New Series), Volume XVII, 1895, page 361.
(3) "A Case of Myxœdema with Symptoms Simulating Ovarian Tumour", *Australian Medical Journal* (New Series), Volume XVIII, 1895, page 277.
(4) "The Question of Safety in Syme's Teaching in Chloroform Anæsthesia", *Intercolonial Medical Journal of Australia*, Volume I, 1896, page 660.
(5) "A Case of Puerperal Septicæmia: Recovery", *Intercolonial Medical Journal of Australia*, Volume I, 1896, page 31.
(6) "Tetanus Successfully Treated by Anti-Tetanus Serum Intravenously Injected", *Intercolonial Medical Journal of Australia*, Volume IV, 1899, page 420.
(7) "The Causation of Death During the Administration of Chloroform", *The British Medical Journal*, April 5, 12 and 19, 1902.
(8) "The Action of Anæsthetic Quantities of Chloroform upon the Blood Vessels of the Bowel and Kidney, with an Account of an Artificial Circulation Apparatus" (with C. J. Martin), *Journal of Physiology*, Volume XXXII, 1905, page 147.
(9) "The Pharmacology of Ethyl Chloride", *Proceedings of the Royal Society, London*, Series B, Volume LXXVIII, 1906, page 391.
(10) "General Anæsthesia in the Light of Recent Pharmacological Research", pamphlet reprinted from "Transactions of the Eighth Session, Australian Medical Congress", 1908.
(11) "A Coroner on Anæsthesia", *Intercolonial Medical Journal*, Volume XIII, page 114. (Letter pointing out that in this journal the credit for his work had been given to others. A case of the prophet in his own country.)
(12) "Relative Value of Chloroform and Ether as General Anæsthetics", *Australian Medical Journal*, Volume XV, 1910, page 109.
(13) "The Action of Ether upon the Circulation", *Biochemical Journal*, Volume V, 1910, page 79.
(14) "Syncope, Collapse and Shock", *Australian Medical Journal* (New Series), Volume I, 1911, page 171.
(15) "Mixed Narcosis", *Australian Medical Journal* (New Series), Volume I, 1912, page 645.
(16) "Chloroform Dosimetry and the Bearing of Some Results of Recent Physiological Research upon the Practice of Anæsthetics", *Australian Medical Journal* (New Series), Volume III, 1914, page 1397; also *International Congress of Medicine*, London, 1913, Part II, 1914, page 121.
(17) "Resuscitation in Threatened Fatalities during Narcosis of General Anæsthetic Agents", "British Association", Section *I*, 1914; also *American Journal of Surgery*, Volume I, 1915, page 69.
(18) "Evidence of Co-Ordinate Action in the Circulatory System", "British Association", Section *I*, 1914.
(19) "The Relation of Ventricular Fibrillation to Clinical Chloroform Syncope" (read before American Association of Anæsthetists, San Francisco, 1915), *The Lancet*, August 7, 1915.
(20) "The Treatment of Asphyxiation in Croupous Pneumonia by Free Breathing", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1915, page 233.
(21) "Formaldehyde in Ether", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1916, page 414.

TWO GROUPS OF ATYPICAL DYSENTERY BACILLI.

By MARGOT MCKIE AND F. M. BURNET.
(*From The Walter and Eliza Hall Institute, Melbourne.*)

IN the course of investigations into the part played by bacteriophage in infantile bacillary dysentery, a number of cases were observed in which no typical Flexner type bacilli were isolated. Most of these, however, showed the presence of non-lactose fer-

menting colonies on the primary plates, and cultures from these were non-motile and fermented the same sugars as Flexner strains. Further investigation showed that some of these strains were of the Sonne type, giving late production of acid in lactose and agglutinating with a Sonne serum.

From the rest two homogeneous groups containing respectively three and nine strains could be separated. The smaller of these obviously represents a Flexner strain, but both serologically and in its phage reactions the type is distinct from the V, W, X, Y, Z types of Andrewes and Inman and from any of the mixed types, such as VZ, which we have isolated. We have referred to this group as Type U. The bacilli falling in the larger group appear to be more distantly related to the true Flexner strains, but for convenience they are spoken of as Type T. The two forms will be discussed separately.

Type U Bacilli.

Type U bacilli were all obtained from typical severe cases of infantile dysentery and showed the biochemical reactions of Flexner strains. The individuality of the group was first recognized by the use of the standard series of phages for preliminary typing according to the method previously described (Burnet, McKie and Wood, 1930). With the six phages in current use the reactions of the Flexner types which we have isolated in the present investigation, are as shown in Table I.

TABLE I.

Reaction of Flexner Types to Standard Series of Phages.

Type.	Phages.					
	3	5	6	20	21	33
T	—	—	—	—	—	—
U	x	x	—	—	—	—
V	x	x	x	—	—	x
W	x	—	—	x	—	x
X	—	x	x	x	x	x
Y	x	x	x	x	x	x
Z	—	x	x	x	x	—
VZ	—	x	x	—	—	—

With this series of phages the reactions of Type U, as of the other types, have been quite uniform.

When isolated, the U strains were agglutinated to about one-fourth titre by an unabsorbed Y serum, but not at 1:100 with absorbed V, W or Z sera. Later tests showed some agglutination by an absorbed W serum. Sera were made against one of the U strains, and the agglutinin content of one of them is shown in Table II, which also includes the significant results from a series of absorption experiments.

It will be seen that Type U bears much the same relation to the "cosmopolitan" types, X and Y, as the three simple types, V, W and Z, do. It may be noted that our stock X strain "Hughes" is agglutinated by a wider range of sera than was the case when it was described by Andrewes and Inman. Both in its phage reactions and serological behaviour it now closely resembles a Y strain.

TABLE II.

Strain.	Serum.					
	U Unabsorbed.	V Absorbed. W, Z.	W Absorbed. V, Z.	X Absorbed. Y.	Z Absorbed. V, W.	U Absorbed. V, Z.
U	2,560	0	400	0	0	1,600
V	480	2,400	0	0	0	0
W	0	0	1,600	0	0	0
X	2,560	150	0	600	200	1,600
Y	2,560	600	400	0	400	1,200
Z	320	0	0	0	1,600	0

Type T Bacilli.

Organisms of Type T were obtained from nine infants admitted as suffering from infantile dysentery. All showed the typical presence of blood and muco-pus in the fæces, but the illness was in most cases less severe than is usual with Flexner infections. There were no deaths in the series, which included two children under six months, and since the mortality amongst proved Flexner cases in the last two years has been around 30% to 40% and is almost 100% in infants under six months, we can almost certainly assume that there is a real difference in the type of disease associated with T strains.

In its general characteristics the organism resembles *Bacillus dysenteriæ* Flexner, being a non-motile Gram-negative bacillus growing well on ordinary media. On MacConkey's agar the colonies are quite similar to Flexner colonies, but on ordinary agar they tend to be rather more opaque and slightly granular in appearance. Growth in broth produces a uniform turbidity.

In Table III are shown the biochemical reactions of the T strains. Except for the late fermentation of dulcite, there are no significant variations from the characteristics of true Flexner strains.

The Bacteriophage Reactions of T Strains.

Throughout the investigations representative colonies of all non-lactose fermenters appearing on the primary plates were tested with a standard series of six dysentery phages according to the technique described previously (Burnet, McKie and Wood, 1930). None of these phages lysed any of the T strains. When a more extensive series of Flexner dysentery phages was examined, two were found to be active against all the T strains. These (numbers 12 and 29) are both small plaque phages of Group N. Further phages acting on the group were readily obtained from pig fæces and other sources, but all showed uniformly small plaques. The nine T strains, when tested with all the available phages, showed almost absolutely uniform reactions (see Table IV). There were only one or two minor differences in the degree of clearing by the active phages. From the point of view of phage reactions, therefore, the group is at least as homogeneous as any of the Flexner races (V, W *et cetera*).

TABLE III.

Biochemical Reactions of T Strains.

Strain.	Glucose.	Lactose.	Mannite.	Dulcite.	Litmus Milk (3 weeks).	Indol.	Methyl Red.	Voges Proskauer.	Catalase.	Acid Agglutination
And.	A	–	A	A (18d)	–	–	–	–	+	–
Bed.	A	–	A	A (18d)	–	–	±	–	+	–
Doolan ..	A	–	A	A (7d)	–	–	–	–	+	–
Hull	A	–	A	A (13d)	–	–	+	–	+	–
Lucas	A	–	A	A (7d)	A.C. (6d)	–	+	–	+	–
Tracey	A	–	A	A (13d)	–	–	+	–	+	–
McCummon ..	A	–	A	A (7d)	–	–	+	–	+	–
Smith	A	–	A	A (13d)	–	–	–	–	+	–
Bawden ..	A	–	A	A (7d)	–	–	+	–	+	–
Bacillus Flexner	A	–	A	–	Sl. A	±	∓	–	+	–

Serological Behaviour.

Emulsion of T strains were agglutinated to a very low titre (40 to 80) by an unabsorbed Flexner Y serum (T 6,400), but were unaffected by V, W, X or Z sera absorbed of all but their specific agglutinins. No agglutination was shown with a Sonne serum nor with a serum agglutinating rough Flexner strains.

The application of the ordinary methods of classifying the dysentery bacilli therefore shows the existence of a homogeneous group of strains closely resembling Flexner bacilli in their biochemical characteristics, but sharply differentiated by their phage reactions and showing little evidence of serological relationship with any of the Andrewes-Inman Flexner types.

Further work on the serological and bacteriophage reactions of the group led to some interesting observations, the significance of which is not yet quite clear.

By the action of phage 53 on any of the T strains, resistant cultures could be obtained which, unlike the parent culture, were agglutinated almost to titre by an absorbed Flexner V serum. Other sera containing the V component, such as unabsorbed X and Y sera, also agglutinated the variant to high titre, and there was slight agglutination (1:160) with an absorbed W serum. This behaviour was uniform throughout the group. Apart from increased agglutinability and a resistance to phage 53 and one or two similar phages, the agglutinable variant seemed to be identical with the parent type. In particular the development of agglutinability was not associated with any increase in sensitivity to dysentery phages. Although agglutinated by a specific V serum, the agglutinable T forms were unaffected by any of the phages lysing true V Flexner strains (Table IV).

Rough variants were derived from each of the T strains and tested with a large series of phages. It will be remembered that there is no type specificity in the phage reactions of dysentery bacilli in the rough phase. All phages of groups C, D, F and N are active, but none of the E (smooth) group. This holds for Shiga and Sonne strains as well as for all the Flexner races.

Rough T strains, however, showed a characteristically different behaviour. Most D, F and N dysentery phages were active as well as those specially developed against the smooth T strains, but none of the C phages had any action. If we tabulate the reactions of typical rough strains, (*a*) of the Salmonella group, (*b*) of the true dysentery bacilli, and (*c*) of the present group, T, against the phage groups, we obtain Table V.

TABLE IV.

Typical Phage Reactions of T Strains and their Variants.

Strain.	Phages.																
	C.			D.			E.				N.				F.	T.	
	5	S18	28	S13	3	4	6	8	21	44	12	13	29	20	48	51	53
Hull (T)	–	–	–	–	–	–	–	–	–	–	±	–	+	–	–	+	+
Hull Va (T)	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	+	–
Hull R (T)	–	–	–	+	+	±	–	–	–	–	+	–	±	±	±	+	+
Flexner V (Smooth)	+	–	–	–	+	+	+	+	–	+	–	–	+	–	+	±	+
Flexner V (Rough)	+	+	+	+	+	+	–	–	–	–	+	+	+	+	+	+	+

TABLE V.

	Phage Type.					
	C.	D.	A and E.	F.	N.	T.
Salmonella R ..	+	±	−	−	−	−
Dysentery R ..	+	+	−	+	+	+
T-R	−	+	−	+	+	+

Typical rough variants were obtained from all the original strains and in addition from all of the agglutinable variants. No difference between the phage reactions of the two types was evident.

The Serological Relationships of the Two Variants.

Sera were prepared by immunizing rabbits with four strains, the original strain and the agglutinable variant (Va) of "Hull" and "Smith". All four sera were identical, as far as could be determined qualitatively, and the reactions of the two "Hull" sera only need be described. Table VI shows the results of agglutinations made with unabsorbed sera, and in Table VII are combined the results of absorption experiments. All the absorptions were made under uniform conditions with equal amounts (opacity determinations) of bacterial emulsion.

TABLE VI.
Agglutination by Unabsorbed T Serum.

Strain.	Serum.	
	Hull.	Hull Va.
Hull T	320	320
Hull Va T	3,840	5,120
Flexner V	320	1,280
Flexner W	<40	40
Flexner X	40	320
Flexner Y	80	320
Flexner Z	<40	<40
Rough V	40	160
Sonne	80	80

TABLE VII.
Reactions of Absorbed T Sera.

Serum.	Strain Agglutinated.		
	Hull (T).	Hull Va (T).	Flexner V.
Hull unabsorbed	320	3,840	320
Hull absorbed Hull	<28	56	28
Hull absorbed Hull Va ..	<28	56	<28
Hull absorbed Flexner V ..	224	3,584	<28
Hull Va unabsorbed	320	5,120	1,280
Hull Va absorbed Hull	<28	224	112
Hull Va absorbed Hull Va ..	<28	28	<28
Hull Va absorbed Flexner V ..	224	3,584	<28

To summarize the serological findings we may say that both variants possess almost identical antigenic structure and approximately equal absorbing power although the original is far less readily agglutinable than the variant. Both possess an antigenic component which is apparently identical with the specific antigen of Flexner V strains, but absorption by Flexner V leaves the titre for the homologous strains practically unaltered. There is some evidence that the Flexner V component is present in larger amount in the agglutinable variant.

Discussion.

For discussing the relationship of these two types of dysentery bacilli to the types described by other workers, the most useful data are to be found in the paper by Kalic (1927), who made a comparative study of the series described by Andrewes and Inman in England, Kruse in Germany, and Aoki in Japan.

None of the strains he describes seems to correspond to our Type U, although the latter may, of course, represent one of the groups of Kruse's series, of which no examples were available, namely, B, C, F and G.

The fact that it is difficult to provoke agglutinins acting against the T strains as isolated brings them close to Kruse's Group I described in 1917. Against this the latter are said to ferment mannite irregularly, while our strains do so in typical rapid fashion.

Kalic describes two strains, Aoki VII and Bristol 15, which differed in important particulars from the typical dysentery bacilli and from our Type T, but, along with the latter, possessed the common feature of being agglutinable to a relatively high titre by V serum.

Conclusion.

Two groups of dysentery bacilli from cases of infantile dysentery in Melbourne are described as races U and T of *Bacillus dysenteriæ* (Flexner).

Acknowledgement.

The cases of dysentery used in our work on bacteriophage at the Children's Hospital were also studied bacteriologically by Dr. W. T. Nelson, whose findings are described in the accompanying paper. We are very grateful to Dr. Nelson for his cordial cooperation in all phases of the work.

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THE SIGNIFICANCE OF "SLOW DULCITE FERMENTERS" IN SUMMER DIARRHŒA.

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THE examination of large numbers of children in Melbourne suffering from diarrhœal disease during the summer months has revealed a type of dysentery

bacilli which, while closely resembling *Bacillus flexneri*, is relatively inagglutinable by a polyvalent agglutinating serum prepared against *Bacillus flexneri*. It also fails to agglutinate with an agglutinating serum of the slow lactose fermenting Sonne type of dysentery bacilli. In addition to its non-agglutination with Flexner and Sonne agglutinating sera, this strain is characterized by the slow fermentation of dulcite in peptone water sugar fermentation tubes.

During the summer of 1930-1931 in Melbourne, 86 out of 122 cases of diarrhœal disease yielded dysentery bacteria which were classified as follows:

Flexner type	71 or 58·2%	of cases investigated
Sonne type	4 or 3·3%	of cases investigated
"Slow dulcite fermenters"	11 or 9·0%	of cases investigated

The "slow dulcite fermenters" were first isolated by us in April, 1928, from a child eleven months old, and were isolated subsequently at various times. An agglutinating serum was prepared from one of the strains, "Haines", from a child of that name suffering from dysentery in the Children's Hospital, Melbourne, in December, 1928.

The cases from which the "slow dulcite fermenters" were isolated presented the following general features:

The ages of these patients lay between three months and two years two months. In a typical case the illness had an acute onset with abdominal pains and diarrhœa. The stools were usually watery, with some mucus and occasionally a marked quantity of pus. Blood was observed at some time in 77% of cases. The presence of blood was not as marked a feature as in the type of case due to Flexner organisms, and usually was observed on only one or two occasions. In two cases the stools were described as being large and of an orange colour. In 82% of cases the temperature charts showed evening rises to only 37·8° C. The duration in hospital averaged twenty-seven days. There were no deaths. Of the patients with dysentery investigated during this season, whose infections were found to be due to the Flexner type of organism, 16·6% died. The general course of the disease in the children from whom the slow dulcite fermenting strains were obtained was, on the whole, less severe than in those due to Flexner dysentery organisms.

Bacteriological Characters of the Slow Dulcite Fermenting Dysentery Bacilli.

Colonies of slow dulcite fermenting dysentery bacilli on agar plates are somewhat larger than those of Flexner dysentery bacilli. They are also more irregular in outline and have a granular surface. The organisms are Gram-negative, non-motile, coliform bacilli. The biological reactions given by these organisms, together with those given by typical examples of *Bacillus flexneri* and *Bacillus alkalescens*, are shown in Table I.

Dulcite Fermentation.

According to Andrewes and Inman:[1]

Flexner's bacillus does not ferment dulcite, but *Bacillus alkalescens* does so. The latter species is also distinguished by the intensity with which it produces alkali in milk and sugar-free proteid media, and by its lack of toxicity.

The organisms described in this paper differ from *Bacillus flexneri* in fermenting dulcite, and from *Bacillus alkalescens* in not producing much alkali in milk and in the fact that they show some toxicity for rabbits and guinea-pigs. The faculty of fermenting dulcite was not observed at first. For routine work dulcite fermentation tests were ordinarily carried out in a medium composed of 1·0% "Difco" proteose peptone, 0·5% dulcite (Merck), 0·5% sodium chloride in distilled water with brom-cresol-purple as the indicator. In this medium dulcite fermentation in the case of *Bacillus alkalescens* is strongly marked usually in less than twenty-four hours, but in the case of the fresh strains of the slow dulcite fermenting organism, acid production was not apparent until after the third day. Acid formation by this group of bacteria, when grown in dulcite peptone water, was subsequently more closely examined. Two sets of media were prepared, one containing 0·5% dulcite and the other 1·0% dulcite, both in 1% "Difco" proteose peptone water with 0·5% sodium chloride.

TABLE I.

Organism.	Motility.	Fermentation.											Litmus Milk.	Indol.	Methyl Red.	Voges Proskauer.	Nitrate Reduction.	Catalase.	Methylene Blue reduction.
		Glucose.	Mannite.	Lactose.	Dulcite.	Saccharose.	Salicin.	Xylose.	Arabinose.	Rhamnose.	Sorbite.	Inosite.							
Slow dulcite fermenting strain, "Lewis" ..	–	A (1)	A (1)	–	A (sl. 5)	–	–	–	A (1)	–	A (sl. 3)	–	sl. A	–	+	–	+	+	+
Bacillus flexneri	–	A (1)	A (1)	–	–	–	–	–	A (1)	–	–	–	sl. A	+	+	–	+	+	+
Bacillus alkalescens ..	–	A (1)	A (1)	–	A (1)	–	–	A (1)	A (1)	A (1)	A (5)	–	Alk.	+	+	–	–	+	+

The figures in parentheses refer to the periods, in days, before acid formation was observed.
– = no reaction, + = positive reaction, sl. = slight acid formation, A = acid formation without gas.

Tubes of these media were inoculated with different strains of dysentery bacteria and incubated at 37° C. for different periods of time. Then the hydrogen-ion concentration of the cultures were determined electrometrically. The plan of this experiment, together with the results, is shown in Tables II and III.

TABLE II.
0·5% *Dulcite*.[1]

Inoculum.	Period of Incubation in Days.		
	1	3	7
	pH	pH	pH
Slow dulcite fermenting strain A	6·9	6·3	6·4
Slow dulcite fermenting strain B	6·9	7·2	6·2
Slow dulcite fermenting strain C	6·9	6·3	6·4
Bacillus flexneri	6·9	7·8	7·8
Bacillus alkalescens	5·4	—	5·4

[1] The initial pH of the medium was 7·2.

TABLE III.
1·0% *Dulcite*.[1]

Inoculum.	Period of Incubation in Days.				
	1	3	7	14	21
	pH	pH	pH	pH	pH
Slow dulcite fermenting strain A ..	5·7	5·7	6·1	6·1	5·6
Slow dulcite fermenting strain B ..	7·0	5·7	6·2	6·2	5·7
Slow dulcite fermenting strain C ..	6·8	5·7	6·0	5·9	5·5
Bacillus flexneri	6·7	7·0	7·7	7·8	7·8+
Bacillus alkalescens	5·2	5·3	5·1	5·4	—

[1] The initial pH of the medium was 7·2.

In order to ascertain whether acid formation would appear in this medium in the absence of dulcite, another batch was prepared, containing 1·0% "Difco" proteose peptone and 0·5% salt in water, and to half of it 1·0% dulcite was added. The media were tubed, inoculated, incubated, and the final reactions determined electrometrically as before (Table IV).

TABLE IV.

Inoculum.	Days of Incubation.		
	1	6	16
	pH	pH	pH
Slow dulcite fermenting strain A in medium with no dulcite	7·2	7·6	7·8
Slow dulcite fermenting strain A in medium with 1% dulcite added	5·8	5·9	5·7
Slow dulcite fermenting strain B in medium with no dulcite..	—	7·7	7·8+
Slow dulcite fermenting strain B in medium with 1% dulcite added	—	5·9	5·8

It would appear that, while 0·5% of dulcite in a "Difco" proteose peptone water medium is adequate to show acid formation when inoculated with a strong dulcite fermenter such as *Bacillus alkalescens*, acid formation is insufficiently shown unless a greater amount of dulcite is used in the diagnosis of "slow dulcite fermenters".

The reactions of dulcite free peptone water cultures of the latter strains of organisms do not become acid under the conditions described above, but they do become acid if 1% dulcite is added previously to inoculation. Dulcite appears, therefore, to have been fermented.

Preliminary Diagnosis of Dysentery Bacilli.

In our work rapid preliminary survey of freshly isolated strains of dysentery bacilli is made by inoculating a suspected colony on the primary plate into the following media:

Indol bouillon (for indol)
Lactose
Glucose
Mannite
Rhamnose } "Sugar" fermentation media

An agar slope is inoculated for further tests.

The inoculated media are incubated for twenty-four hours at 37° C. The cultures are then examined for fermentation of lactose, and the non-lactose fermenters are examined further for changes in the medium containing glucose. Those which do not ferment lactose, but form acid in glucose, are examined for motility and reaction to the Gram stain, the material for these tests being derived from the indol bouillon. Should an organism be found to be Gram-negative and non-motile, it is then classified according to the following plan as a guide for further tests (Table V).

Indol Formation.—Although there is apparently some doubt as to whether all strains of *Bacillus flexneri* form indol, it was found that all strains of this organism isolated by us in Melbourne did form indol, whereas the Sonne and slow dulcite fermenting strains of dysentery bacilli did not. The medium in routine use at the Commonwealth Serum Laboratories for testing for indol formation is the "indol bouillon" of Kristensen, Lester and Jürgens.[2] The method of testing for the presence of indol is that described by Topley and Wilson.[3]

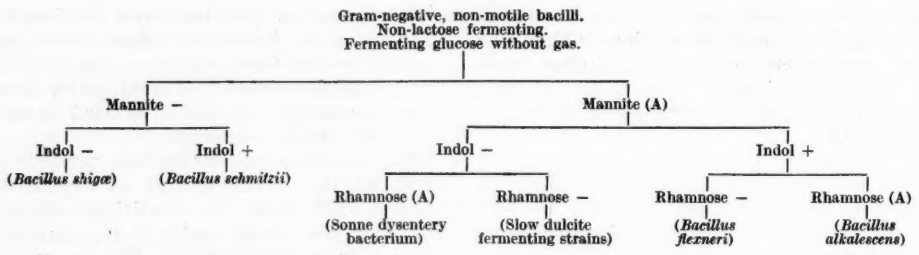
Rhamnose Medium.—Rhamnose, or "isodulcite", is useful because it is not readily fermented by *Bacillus flexneri* nor by the slow dulcite fermenting strains, whereas it is fermented in twenty-four hours by the Sonne organism and by *Bacillus alkalescens*.

A preliminary classification having been made according to the above scheme, confirmatory evidence is obtained by further fermentation tests, such as late fermentation of lactose and of dulcite, and by agglutination tests.

Serological Relationships of the Slow Dulcite Fermenting Strains with Bacillus Flexneri.

Agglutinating sera were prepared by immunizing four rabbits, each with a separate strain of this

TABLE V.



group. The tests for agglutination and absorption of agglutinins showed these strains to be identical, thus forming a homogeneous group. It was found difficult to produce a high titre serum in rabbits by inoculating with "slow dulcite fermenters". Likewise, patients' sera would not agglutinate the homologous organisms to a high titre. In these respects they resemble *Bacillus alkalescens*.

Agglutination Reactions with Flexner Agglutinating Sera.

The dulcite fermenting strains are agglutinated by polyvalent Flexner serum (V, W, X, Y and Z) to a titre of only one in 80 or one in 160, being about 6% of the titre for the various strains of *Bacillus flexneri*.

Table VI shows the extent of their agglutination by monovalent *Bacillus flexneri* agglutinating sera in a saline medium buffered at pH 7·0 according to the method of A. C. Evans.[4]

Absorption of Agglutinins from Monovalent Bacillus Flexneri Agglutinating Sera by a Slow Dulcite Fermenting Strain of Bacteria.

The absorbing dose used in each case amounted to about 500,000 million organisms per cubic centimetre of undiluted serum. The residual titres of the absorbed sera and the titres of control sera are shown in Table VIII.

Absorption of Agglutinins from "Slow Dulcite Fermenting" "Haines" Serum by Strains of Bacillus Flexneri.

The absorbing dose used in absorption of agglutinins from "slow dulcite fermenting" "Haines" serum by strains of *Bacillus flexneri* was that described above. The results are shown in Table IX.

These results show that while the dulcite fermenting strain has something in common antigenically with *Bacillus flexneri*, yet it is distinct from it. It appears to be more nearly related to the Y type of *Bacillus flexneri*.

Evidence of Pathogenicity.

"Slow dulcite fermenters" have been found in practically pure culture in the primary plating of muco-pus from the stools of patients. In those cases from the stools of which this type of bacterium has been isolated, no other organism of an accepted type of pathogenic dysentery bacillus was found to coexist. On the other hand, organisms of doubtful

TABLE VI.

Agglutinating Reactions of Slow Dulcite Fermenting Strains by Monovalent Bacillus flexneri Agglutinating Sera in Buffered Saline Solutions.

Serum.	Dilutions of Serum.								Control.	Percentage titre of agglutination.
	1/40	1/80	1/160	1/320	1/640	1/1280	1/2560	1/5120		
Bacillus flexneri V *versus* V serum	+++	+++	+++	+++	+++	+++	+++	++	−	
"Slow dulcite fermenter," "Bedella" *versus* V serum	+++	+	−	−	−	−	−	−	−	1·5
"Slow dulcite fermenter," "James" *versus* V serum	+++	+	−	−	−	−	−	−	−	1·5
Bacillus flexneri W *versus* W serum	+++	+++	+++	+++	+++	+++	+++	−	−	
"Bedella" *versus* W serum	−	−	−	−	−	−	−	−	−	—
"James" *versus* W serum	+	−	−	−	−	−	−	−	−	1·5
Bacillus flexneri X *versus* X serum	+++	+++	+++	+++	+++	+++	−	−	−	
"Bedella" *versus* X serum	++	−	−	−	−	−	−	−	−	2·0
"James" *versus* X serum	++	−	−	−	−	−	−	−	−	2·0
Bacillus flexneri Y *versus* Y serum	+++	+++	+++	+++	+++	+++	++	−	−	
"Bedella" *versus* Y serum	+++	+++	++	−	−	−	−	−	−	6·2
"James" *versus* Y serum	+++	+++	++	−	−	−	−	−	−	6·2
Bacillus flexneri Z *versus* Z serum	+++	+++	+++	+++	+++	+++	++	−	−	
"Bedella" *versus* Z serum	+	−	−	−	−	−	−	−	−	1·5
"James" *versus* Z serum	+	−	−	−	−	−	−	−	−	1·5

TABLE VII.

Agglutinating Reactions of *Bacillus flexneri* with Slow Dulcite Fermenting "Haines" Serum in Buffered Saline Solution at pH 7·0.

Serum.	Final dilution at which agglutination occurred.	Percentage of titre of "Haines" serum at which agglutination occurs.
"Haines" serum *versus Bacillus flexneri* V organisms	1 in 480	25
"Haines" serum *versus Bacillus flexneri* W organisms	1 in 240	12·5
"Haines" serum *versus Bacillus flexneri* X organisms	0	0
"Haines" serum *versus Bacillus flexneri* Y organisms	1 in 480	25
"Haines" serum *versus Bacillus flexneri* Z organisms	1 in 240	12·5
"Haines" serum *versus* Bacillus "Haines"	1 in 1920	

TABLE VIII.

Serum and Organisms.	Dilution.
Bacillus flexneri V serum absorbed by "slow dulcite fermenter" *versus* V organisms	1 in 7,680
Bacillus flexneri V serum control	1 in 7,680
Bacillus flexneri W serum absorbed by "slow dulcite fermenter" *versus* W organisms	1 in 1,920
Bacillus flexneri W serum control	1 in 1,920
Bacillus flexneri X serum absorbed by "slow dulcite fermenter" *versus* X organisms	1 in 1,920
Bacillus flexneri X serum control	1 in 1,920
Bacillus flexneri Y serum absorbed by "slow dulcite fermenter" *versus* Y organisms	1 in 1,920
Bacillus flexneri Y serum control	1 in 1,920
Bacillus flexneri Z serum absorbed by "slow dulcite fermenter" *versus* Z organisms	1 in 1,920
Bacillus flexneri Z serum control	1 in 1,920

TABLE IX.

Showing Final Dilutions of Sera at which Agglutination was Observed.

Serum and Organisms.	Dilution.
"Haines" serum absorbed by *Bacillus flexneri* V *versus* "Haines" organisms	1 in 1,920
"Haines" serum absorbed by *Bacillus flexneri* W *versus* "Haines" organisms	1 in 1,920
"Haines" serum absorbed by *Bacillus flexneri* X *versus* "Haines" organisms	1 in 1,920
"Haines" serum absorbed by *Bacillus flexneri* Y *versus* "Haines" organisms	1 in 1,920
"Haines" serum absorbed by *Bacillus flexneri* Z *versus* "Haines" organisms	1 in 3,840
"Haines" serum control	1 in 1,920

pathogenicity, such as *Bacillus morgani* number 1 and *Bacillus alkalescens*, are commonly found in the stools of patients together with *Bacillus flexneri*. The type of illness affecting patients from whom "slow dulcite fermenters" were isolated was generally a milder form of dysentery than that caused by *Bacillus flexneri* and was not accompanied by any mortality.

Agglutination Tests of Patients' Sera.

Blood sera of three patients from whom "slow dulcite fermenters" were isolated were found to agglutinate the organisms in dilutions up to one in 40.

As controls, the blood sera of six children of ages corresponding to those of the patients agglutinating slow dulcite fermenting organisms were tested for the presence of agglutinins for these organisms. No agglutination was found in dilutions of sera from one to ten upwards.

Animal Toxicity Tests.

Eighteen-hour agar slope cultures of three different strains of the slow dulcite fermenting species were each emulsified in three cubic centimetres of 0·85% saline solution and used for intravenous injection into "English" rabbits in accordance with the following plan: Rabbit A received whole slope, strain "Haines", and marked diarrhœa resulted. Rabbit B received whole slope, strain "Dennis"; death occurred two days later. *Post mortem* general peritonitis was present. The organism was recovered in almost pure culture from the small bowel. Rabbit C received half slope, strain "Anderson"; there was no diarrhœa and no apparent ill effects followed.

Eighteen-hour agar slope cultures of three different strains were used, one-quarter of each slope being emulsified in saline solution and injected intraperitoneally into three guinea-pigs respectively. The guinea-pigs all died, one after twenty-four hours and the other two after forty-eight hours.

"Atypical *Bacillus dysenteriæ*" with biological reactions closely resembling those described in this paper were found by T. J. Mackie[5] in Egypt. Similar organisms are described by Mackie and McCartney.[6] The writer has little doubt that these "slow dulcite fermenters" are the ætiological agents responsible for certain cases of dysentery in Melbourne. Whether they should be included amongst the Flexner group is, however, open to doubt. They have certain definite and apparently fixed characteristics which enable one to distinguish them from the Flexner types.

Summary.

A strain of dysentery bacilli is described resembling a race of "atypical *Bacillus dysenteriæ*" of Mackie and McCartney[6] in being a slow fermenter of dulcite. It has been found repeatedly since 1928 in the stools of children suffering from diarrhœal diseases. During the summer of 1930-1931 it was isolated from the stools of 9% of one hundred and twenty-two patients with diarrhœal disease examined in Melbourne. This strain was found to resemble *Bacillus flexneri* in many ways, but differed from them in the following respects: (*a*) The appearance of the colonies on agar plates, (*b*) fermentation of dulcite, (*c*) no formation of indol, (*d*) serological reactions (agglutination and absorption of agglutinins).

The type of illness affecting the children in whom it was found was a milder form of dysentery than that caused by *Bacillus flexneri*, for there were no deaths; while of the patients suffering from dysentery due to *Bacillus flexneri*, 16·6% died. Pathogenicity for human beings tends to be confirmed by the development of agglutinins in patients' sera and by the absence of the well recognized types of dysentery bacilli from the stools. Some evidence of pathogenicity for laboratory test animals is given.

Acknowledgements.

It was found that Dr. F. M. Burnet and Miss M. McKie, working together, and the writer, independently, had isolated similar organisms from the same group of cases in the summer of 1930-1931. I am greatly indebted to Dr. Burnet and Miss McKie, who very kindly delayed publication to allow our papers to appear together.

I desire to thank Dr. F. G. Morgan, Director, Commonwealth Serum Laboratories, Department of Health, for his interest and encouragement, and Mr. F. J. Considine for helpful suggestions and for carrying out the pH determinations.

I am indebted to Mr. Ivan Stephens and to Mr. C. V. Vaughan, B.C.E., for much of the routine work in connexion with this investigation.

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RADIUM IMPLANTATION IN THE TREATMENT OF CAPILLARY AND CAVERNOUS NÆVI.

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RADIUM has long been recognized as one of the most favourable means of treating nævi. The customary method of application, by means of surface applicators, has been very successful, but it is unusual to obtain a satisfactory result with one treatment. By a satisfactory result is meant the disappearance of the tumour without marked reaction and without leaving any apparent scarring.

Being dissatisfied with some of the results obtained by surface applications (plaques), I experimented with different methods of applying radium. I secured satisfactory results, following the suggestions of an American author, by using radium at a distance of one centimetre; but, like other surface methods of attack, this required a number of applications. In the endeavour to reduce the number of treatments necessary without producing an excessive reaction, intratumoral application was attempted with very satisfactory results. The idea of implantation was gained from electrocoagulation of these growths where, of course, one employs a special needle with an insulated band for protecting the transfixed skin from damage.

The method does not always appear to be very satisfactory in large subcutaneous nævi, unless the whole of the growth is simultaneously transfixed with needles. Where this cannot be done, it would appear that better results will be obtained in very large cavernous nævi by injection treatment, such as is used for varicose veins. (Personal communication from Dr. T. Fergus.)

In all, six patients with seven nævi were considered suitable for implantation therapy during the period under review. Four of these gave a perfect and immediate result, two were very greatly improved, and one nævus which did not respond to implantation, gave a satisfactory result with surface applications.

Technique.

Except for the unsuccessful case mentioned above, steel needles, 29 millimetres in length, with walls 0·3 millimetre thick, containing 10 milligrammes of radium element, were used. In the unsuccessful case platinum needles 22 millimetres long, with 0·3 millimetre screenage, containing five milligrammes of radium element, were employed.

The skin overlying the tumour was first cleansed and rendered aseptic with rectified spirit, following which the sterilized needles were inserted to produce an even irradiation throughout the growth, and remained *in situ* for two and a half to three hours. The needles were then withdrawn and the wounds sealed with collodion.

Case Reports.

CASE I.—Baby P., aged ten weeks, had a large rounded mass on the right shoulder. The mass originally was small and pale, but had increased in size rapidly and become deeply coloured. The deeper subcutaneous portion of the mass was 30 millimetres in diameter and the superficial 15 millimetres, while the total depth was 15 millimetres.

Treatment was carried out by inserting three five-milligramme needles radially into the base of the mass for two and a half hours on July 9, 1928. Some improvement followed this, but by August 20 the mass was as large as ever. This time four needles were inserted radially as before for three hours. Again only a temporary improvement followed. It was obvious from this that the duration of treatment was insufficient, but continued anæsthetics were considered undesirable and surface treatment was instituted. Between October 17, 1928, and July 26, 1929, seven applications of radium at a distance of one centimetre were made, and on March 12, 1931, the mother reported that there was no sign of the growth, only a pink mark on the skin.

CASE II.—Elsie R., aged three months, had a rapidly growing capillary nævus of the hairy scalp above the right ear. The nævus was bright red, slightly raised, and involved the whole thickness of the scalp. Its area was 14 by 12 millimetres, and, as I considered that surface applications would probably cause a loss of hair, I advised implantation of radium. This was carried out, one ten-milligramme needle being inserted for three hours. Two months later the only sign of the previous growth was a slightly depressed scar, and there had been no loss of hair.

CASE II.—Robert A., aged five months, had a rapidly growing bright red capillary nævus, somewhat irregular in outline and roughly 20 by 10 millimetres, in the centre of his back. Two ten-milligramme needles were inserted on December 10, 1929, and twelve months later his father, a medical man, reported there was no evidence of the former growth.

CASE IV.—Doreen A., aged five m nths, had a rounded, reddish tumour in the upper lip, immediately to the right of the middle line. The mass was 15 millimetres in

diameter and considerably raised, but did not involve the whole thickness of the lip. This was a difficult case to treat, owing to the difficulty in retaining an applicator in place. On December 17, 1929, the base of the tumour was transfixed with a ten-milligramme needle inserted from the buccal surface of the lip for three hours. Unfortunately the needle was not inserted deeply enough and an incomplete result was obtained. The mass was reduced in size to ten millimetres diameter, and was paler. Several surface applications were carried out subsequently with further improvement, but the child has not been seen for a year.

CASE V.—Shirley G., aged three months, was seen on April 4, 1930, when she had a large subcutaneous nævus in the neck with involvement of the overlying skin. The size of the mass was 27 by 21 millimetres, and there was a further small nævus on the hairy scalp. When she cried, the mass in the neck increased greatly in size.

Four ten-milligramme needles were inserted into the cervical growth for three hours on the occasion of her first visit. Two months later there was not much improvement in the cervical mass, and the scalp mass was larger. Three ten-milligramme needles were inserted in the cervical growth and one into the scalp tumour for a further three hours.

Ten weeks later the scalp tumour had disappeared, as had the subcutaneous portion of the growth in the neck, though the cutaneous discoloration persisted. This was somewhat similar to, but not exactly typical of a *nævus flammœus*. It is showing only a slight further improvement with surface applications.

CASE VI.—Prudence S., at seven weeks, came with a large subcutaneous mass overlying the right shoulder blade. It had been discovered some two weeks previously following surgical removal of a rapidly growing cutaneous nævus from a leg. In the meantime it had increased rapidly in size, being 30 by 20 millimetres, and gave the overlying skin a peculiar dull white appearance.

Three ten-milligramme needles were inserted for three hours on August 26, 1930, and six months later the growth had completely disappeared.

Discussion.

For Case I, five-milligramme needles screened with 0·3 millimetre platinum were used. This was largely an experiment, and I considered it desirable to feel my way gently. The immediate result was such as to indicate the possibility of the method; but owing to the number of anæsthetics required and to the production of slight puckered scars, I considered it undesirable to experiment further with this patient (a girl). It is just possible that the radial distribution of the needles allowed the central vessels to escape and so undid any good result obtained at the periphery. The effect would be the same as with the central dilated vein of a stellate nævus, which it is useless to treat with radium. Inspection of tables in Bachem ("Principles of X-Ray and Radium Dosage") and Colwell and Russ ("Radium, X-Rays and the Living Cell") shows that the relative screening power of equal thicknesses of platinum and iron is approximately as three to one, so that the more probable explanation of our failure in this case, in the light of later cases, is that the period of treatment was unduly short.

When the second patient (Case II) appeared on the scene, I decided to use an applicator with less screenage, namely, 0·3 millimetre steel. This certainly had the desired effect, and I feel that the β radiation probably intensified the effect.

In Case IV I attribute the unsuccessful result to the fact that my applicator was too large and clumsy to allow accurate transfixation of the growth and I feel sure that had I available a smaller applicator, a completely satisfactory result would have been obtained here.

The partially unsuccessful result obtained in Case V is possibly due to the likeness of the superficial portion of the growth to a *nævus flammœus*, and perhaps also to an unnoticed error in technique.

Of course, in these modern times it is difficult to obtain applicators such as I have indicated, though there is no reason why radon should not replace radium element. When heavier screening, which I consider unnecessary in these cases, is employed, the period of treatment requires to be correspondingly prolonged.

No trouble was experienced through sepsis.

THE IMPORTANCE OF DRAINAGE IN RENAL DISEASE.[1]

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INFECTIONS of the kidney run a course which is influenced by several different factors. In some cases the course run is quite short and the recovery appears to be complete after a few weeks. In other cases patients are often seen who run indefinitely with chronic pyelitis, and no form of treatment appears to influence them in any way. This inconsistency depends on the different elements producing the condition.

The three chief factors which influence the course of renal infection are: (i) The type of organism infecting the kidney; (ii) the efficiency of the renal drainage; (iii) the presence elsewhere in the body of focal sepsis which is causing continuous reinfection of the kidney.

Infections of the kidney may be due to a number of different organisms, which may affect it in different ways, but in this particular case we shall deal with the more common non-tuberculous infections. These infections, which are generally due to *Bacillus coli*, staphylococci or streptococci, may be divided into acute and chronic forms. The acute form of the disease is termed pyelonephritis, while the chronic condition is generally termed pyelitis. The term pyelitis in either the acute or chronic sense is really a misnomer, as in practically every case of pelvic infection there is a considerable involvement of the parenchyma, so that the term pyelonephritis is really a more accurate one. In dealing with renal infections the first thing to be clearly borne in mind is that kidney infections are not primary, but secondary. Organisms are not suddenly generated in the kidney, but reach it generally by the hæmatogenous route after having

[1] Read at the second annual reunion of the Residents' and Ex-Residents' Association, Royal Prince Alfred Hospital, Sydney, October, 1931.

orginated from some focus of infection elsewhere in the body. As these focal infections can frequently be demonstrated, a careful search should always be made for them, and any suppurative process which may be discovered should be energetically attacked and, if possible, completely cleared up. These foci are generally found in the teeth, tonsils, sinuses or gastro-intestinal tract. The teeth and tonsils are the commonest offenders, which is somewhat fortunate, as they are the easiest to remove.

While *Bacillus coli* is by far the commonest organism found in renal infections, especially in the more chronic cases, it would seem that the streptococcus is the one which causes the initial lesion. Once the streptococcus has set up a pyelonephritis, the ever-present and more hardy *Bacillus coli* gains entrance, and, as the lesion progresses, the streptococcus is gradually eliminated and the colon bacillus becomes established.

Bumpus and Meisser, of the Mayo Clinic, did some very illuminating work on the bacteriology of pyelitis and pyelonephritis. They injected a series of eighty-two rabbits with streptococci obtained from foci in the teeth and blood of patients suffering from pyelonephritis, and in sixty-three animals of the series they were able to produce lesions of the kidneys. They believe that this is definite evidence that pyelonephritis originates from focal infections harbouring streptococci, which have a selective affinity for the urinary tract. They also showed that the colon bacillus, which is so commonly found in lesions of this type, is really a secondary invader.

In treating focal sepsis it is the streptococcal variety that must be most severely attacked, and of these areas the intestinal foci are the most difficult to demonstrate and the most inaccessible when it comes to eradication. Infection generally reaches the kidney by the hæmatogenous route, but there are various other ways by which it may get there. Organisms may be carried directly to the kidney by means of a ureteral catheter. Some of the lymphatic channels from the intestine enter the same retroperitoneal lumbar lymphatic glands, as do some of the channels from the kidney. Infection may be possible by this route, but this has never been actually proved. Since the secretory stream carries the products of inflammation to parts at a distance, descending infections are the rule. Any site below the original lesion may be affected, local predisposition playing a part. For instance, in pyelitis the trigone and posterior part of the urethra may be affected while the fundus of the bladder is normal. A number of observers contend that pyelitis is always preceded by a lesion of the parenchyma. Controversy has raged for ages over the mechanism of ascending infection. Infection may spread in a direction opposed to the secretory stream in three ways: (*a*) by the lumen, (*b*) by the lymphatics, (*c*) by the blood stream. Infection by the lumen cannot occur unless there is reverse peristalsis, obstruction to the flow, or unless there is an increase of pressure below, which forces the contents back passively, that is reflux.

Ascending infection by the lymphatics has been demonstrated experimentally in some interesting work done by Sweet and Stewart. They showed that if the outside of the ureters was exposed to infection without opening into the lumen, diffuse infection of the kidney resulted, with little or no infection of the ureteral mucosa. When a piece of rubber tubing was substituted for the middle portion of the ureter, the lymphatic channels, but not the lumen, being thereby interrupted, infection did not ascend to the kidney. According to these observers, ascending infection of the kidney does not occur unless the bladder wall is invaded in the neighbourhood of the ureteral orifices.

In regard to infection by way of the blood stream: an infection of the lower tract can reach the upper tract only through the general circulation. Although there are anastomoses between the blood vessels of the various urogenital systems, the currents do not ascend. There are blood channels all the way from the bladder to the kidney, but the flow is in the opposite direction. No blood passes directly from the bladder to the kidney, except through the *vena cava* first and thence to the kidney by way of the renal artery.

The resistance of the normal kidney to infection is remarkable when one considers that it is the filter for the entire blood stream. The endless bombardment it must get with organisms without becoming in any way infected, is surprising. For instance, in cases of typhoid fever the typhoid bacilli seem able to penetrate the kidney without causing any definite or permanent lesion. The same state of affairs is frequently found in pneumonia. Grawitz did some interesting work along this line by demonstrating that mould spores appeared in the urine after intravenous injection. Finally, taking into consideration the numerous cases of positive blood cultures which are seen without any renal involvement, it is a striking demonstration of the resistance of the normal kidney.

However, in cases where infection of the kidney has actually occurred and become well established, its resistance may be seen in a different way, that is, its resistance to all forms of treatment, which is what chiefly concerns us in this article.

In acute cases of pyelonephritis rest in bed and abundant water are necessary. Urinary antiseptics can be tried, but are of doubtful value. Large doses of alkali often help. The essential thing is to look for and eliminate any primary focus which may have caused the condition. If this is done, the pyelonephritis will frequently run its course and then clear up, but if it fails to respond after a moderate course of ordinary treatment, it is not much use persisting with this type of treatment. When this occurs, there is undoubtedly some other condition complicating the pyelitis.

The most important part of the treatment of the ordinary non-tuberculous kidney infection is free drainage. It is almost impossible to clear up these cases unless good drainage is established. The commonest cause of defective drainage is the presence

of some obstructive lesion, and of these emphasis has been placed on such conditions as body form, sagging kidney, aberrant vessels, ureteral kinks, presence of stone, various intrarenal and extrarenal, also intraureteral and extraureteral lesions, tumours, inflammatory masses, disturbances of neuro-muscular rhythm, pregnancy *et cetera*. Any of these conditions complicating a pyelitis may cause defective drainage and keep the infection going indefinitely, if not recognized. In addition, renal lesions are often silent and therefore not recognized. For instance, a renal stone which is not causing acute obstruction may reach very large dimensions without causing physical symptoms, but no treatment will have any effect on the marked pyuria unless the presence of the stone is recognized. Defective drainage may be the result of a congenital abnormality, such as a displaced kidney, the high insertion of the ureter into the pelvis or it may be mechanical, such as the presence of stone, aberrant blood vessels, spasm of the peristaltic wave, or ureteral stricture. Any of the foregoing conditions will lead to defective renal drainage and will greatly retard or may actually prevent the clearing up of an infection, so that further treatment must be instigated to establish better drainage.

In the case of calculus, especially if it is causing any obstruction, removal is essential, both in order to clear up the infection and also to preserve the renal function. The presence of a calculus which is causing any degree of obstruction in an infected kidney is also causing considerable damage to its function.

When the defective drainage is due to congenital abnormality, such as horseshoe kidney, ectopic kidney or to abnormal insertion of the ureter into the renal pelvis, the establishment of better drainage is going to be much more difficult and in many cases impossible. Under these conditions it will just have to be accepted.

When the obstruction is extrarenal or extraureteral, such as aberrant blood vessels, tumour or inflammatory masses, fibrous bands causing a kinking of the ureter *et cetera*, these may all be rectified by operative interference. Simultaneously with the removal of the obstructive element the kidney can be placed in such a position that the drainage is improved.

In cases of disturbed neuro-muscular rhythm, the peristaltic wave, instead of flowing in an even manner from the pelvis down the ureter, gets disturbed and results in spasm at the uretero-pelvic junction, and this leads to obstruction, dilatation and consequent bad drainage. This type of condition is extremely difficult to treat by any non-operative measures, as the cause of the disturbance in the neuro-muscular rhythm or peristaltic wave cannot be determined, so that the condition generally persists in spite of any treatment. The benefits to be derived from operative interference are also questionable. At operation some cause for the disturbance in the peristaltic rhythm may be discovered and removed, but in most cases the actual cause of this abnormality cannot be determined. The possibility of improving conditions by interfering with the sympathetic nerve supply has to be considered; there has been considerable research and controversy on the advisability of this procedure. In the first place one has to be absolutely certain that the obstruction at the uretero-pelvic junction is entirely due to a spasm of the peristaltic wave at that point. This may represent only one factor in the case; if so, the benefit to be obtained from cutting off the nerve supply is very questionable. In many cases this procedure would appear to be very undesirable and contraindicated. In the event of there being a moderate degree of hydronephrotic development resulting from a persistence of the spasm over a considerable period, an actual stricture formation may have taken place. Fibrous tissue becomes formed, making the contracture permanent and beyond the stage when any relaxation of the nervous spasm will have any beneficial effect. The removal of the nerve supply now merely results in a loss of tone in the musculature of the renal pelvis and ureter while the obstructive factor still persists. Hydronephrotic development would seem to be aggravated rather than checked or improved in such cases.

In some conditions in which the trouble is unilateral, such as a badly infected hydronephrosis with a normal, good functioning kidney on the other side, it is no good persisting with conservative treatment; nephrectomy is indicated early, before the good kidney has become subject to toxic nephritis.

Ureteral stricture may be the cause of bad drainage, and without the passage of a ureteral catheter it may be impossible to discover it, but it is enough to cause a pyelitis to persist indefinitely and to resist all forms of treatment. Until the stricture is overcome by the passage of ureteral catheters and free drainage is reestablished, there is no chance of the infection clearing up.

In the foregoing conditions there have been instances of renal infection complicated by defective drainage, and in fact almost universally this is the underlying cause in conditions of persistent non-tuberculous kidney infections. No urinary infection is likely to be cleaned up while such a state of affairs persists, and for that reason a full urological investigation of these patients should be made and the underlying cause of the condition discovered if possible. Hunner, of the Johns Hopkins Hospital, considers that the mere establishment of good drainage in cases of pyelitis complicated by ureteral stricture is enough to cure the condition. He even goes so far as to say that pelvic lavage is unnecessary if good and effective drainage is obtained.

In some cases this may hold good, but in every instance an attempt should be made to determine the cause. It is possibly because of the passage of ureteral catheters and the consequent dilatation of the ureters rather than because of the actual antiseptics employed that such satisfactory results follow pelvic lavage.

It can be seen that the conditions which may give rise to impaired renal drainage are numerous, so that in the presence of chronic infection an attempt should always be made to determine the underlying cause. If one is discovered, measures must be taken to rectify it, as a urinary infection is not likely to be improved where complicated by defective drainage.

AFTER-RESULTS OF THE ETHMOID OPERATION.

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WHILE doing post-graduate work in the Hajek Clinic in Vienna some time ago, I was struck with the number of patients whom I saw complaining of almost persistent headaches following a complete ethmoid operation. These people had been well operated upon, they had healed and epithelialized cavities, often no discharge, and yet had headaches of a persistent type. In many cases this was apparently not complained of by the patient before the operation. Further, many of these patients were particularly shown to us to impress us with the often found bad after-result of an apparently good operation. I asked what percentage got headaches, and the answer was: "Well, we have not taken statistics, but I should say 50%." This made me decide to circularize the first batch of patients I could find and inquire how they were. As a result I sent a *questionnaire* to patients whose cards I could find, who had been operated upon in the Kent County Ophthalmic and Aural Hospital, Maidstone, since 1924, and although over eighty letters were sent out and I received only twenty-six replies, I feel the trouble to obtain even this number makes their publication worth while and may arouse an interesting discussion.

The *questionnaire*, after explaining my mission, presented the following nine questions. In order to find out why the patient sought medical advice the first was:

Question: Of what did you complain when you came for treatment?

Answer: Two patients were dead. One had died from meningitis within three weeks of the operation and the other died from other causes one year later, at the age of seventy.

The replies were as follows:

Headache and nasal discharge	9
Headache, nasal discharge and obstruction	5
Headaches	3
Headache and failing vision	2
Headache and nasal obstruction	1
Asthma	2
Recurrent conjunctivitis	1
Dilated pupil	1

Question: Are you now free from that complaint?

Answer: Eleven state that they are free, one is partially free, and twelve are the same.

Question: Did you have discharge from the front or back of the nose?

Answer: Seventeen did and seven did not.

Question: Have you got discharge now?

Answer: Twelve have, one has occasional discharge and eleven have not.

Question: Did you have pain or headache before your operation and, if so, where was it located?

Answer: Twenty had and four had not. Of the twenty who had headaches, although the patient replied "over the eye", "bridge of the nose", "bridge *et cetera*", fourteen may be grouped as naso-frontal headaches, one as pain in the side of the head, four as pain in the back of the head, and one simply answered, "headaches".

Question: Have you pain or headache now?

Answer: Five have occasional headaches, in three the headaches are the same, and one has a headache which he did not have before the operation. Therefore, eleven out of twenty with preexisting headaches are cured or partially relieved, and one has acquired a headache apparently as a result of the operation.

Question: Is your general health better or worse than before your operation?

Answer: Fourteen say that it is better and ten say that it is not.

Question: Did your nose seem blocked before the operation?

Answer: Fourteen say yes and ten say no.

Question: Does it seem blocked now?

Answer: Seven say yes and seventeen no.

When we analyse these figures, we find the following: First, the most common complaint is headache. It was complained of by 83% of total sufferers, and was cured in 55%. On the other hand, it was cured in 60% of those who complained of naso-frontal headaches, and much relieved in a further 30%. Only 10% remained the same. In those who complained of headache in the side of the head or occipital area, only 50% were cured. Five *per centum* acquired a headache apparently as a result of the operation.

The next most common complaint was nasal discharge, 71% of cases. Of these 46% still have their discharge, therefore it was cured in only 47% of cases.

The next most common complaint was nasal obstruction, 67%. It is still present, or at least complained of, although perhaps not really present, in 50% or, in other words, was cured in only 25%.

The general health of 62% is better than before their operation. Of the two who complained of failing vision amongst other things, one is the same and the other is worse. The patient who had asthma still has it, and the one who complained of dilated pupil is cured.

Summary.

When we remember that the real object of surgery is to relieve those symptoms from which the patient is suffering, we may say that: (i) When the operation is done for the relief of naso-frontal headaches, it is a good one and has a good chance of success, 90% of patients being cured or relieved, while when done in the presence of headaches elsewhere, it has only a moderate chance of success. (ii) When the operation is done for the relief of nasal discharge, the chances of success are only moderate. (iii) When the operation is done in the hope of relieving a nasal obstruction, the chance of success is indeed remote.

The patient with dilated pupil and recurrent conjunctivitis was cured and the patients with asthma and failing vision were not cured. These last are too small in number upon which to make any comment and are recorded only to make the list complete.

Reports of Cases.

TWO CASES OF TORSION OF AN APPENDIX EPIPLOICA.

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TORSION of the *appendix epiploica* of the colon seems to be comparatively rare, and its ætiology and symptomatology are still sufficiently obscure to warrant the reporting of cases.

I believe that many more cases occur than are recorded, yet in 1927 Johanssen, according to Colt,[1] was able to collect only twenty cases from the literature. Since then several more have been reported, notably three by G. H. Colt,[1] whose specimens are catalogued in the Royal College of Surgeons Museum.

The two following cases occurred in my hospital practice within three months of one another.

Case I.

C.A., a married woman, aged fifty-two, was admitted late one evening, complaining of pain in the right side which had commenced at 11 a.m. the same day. She had vomited once and the pain had persisted all day. The only other item in her history of any importance was that for three days prior to the onset of pain she had been suffering from a severe cold and a hacking cough.

On examination she presented the typical clinical picture usually associated with an acute appendicitis, namely, a temperature of 38° C. (100·4° F.), a pulse rate of 110 and tenderness and slight rigidity on palpation over the right iliac region. Her abdomen was very fat and pendulous. A few scattered râles were audible over the back of the chest.

The condition was diagnosed as acute appendicitis, and operation was undertaken through the usual muscle-splitting incision. The appendix was normal, except for a certain amount of congestion of the vessels. This extended over the peritoneal coat and became more intense as it was traced towards the caecum. At the back of the caecum and directly opposite the base of the vermiform appendix a twisted *appendix epiploica* was found, purple black in colour and twisted tightly three times at its junction with the colon. It was three centimetres in length. No other lesion was found in the abdominal cavity. The offending *appendix epiploica* and the vermiform appendix were both removed and the wound closed. The patient made a recovery which was uneventful, except for some discomfort due to the coexistent lung condition.

Case II.

L.A., a business man, aged fifty-eight, was admitted on January 3. Two nights prior to admission he had been seized with an acute pain in the left lower part of the abdomen, necessitating the administration of morphine for its relief. The pain was attributed to a renal colic. He took castor oil in the morning with no result. His pain came on again during the day and at intervals during the night. He commenced vomiting the next morning and vomited intermittently until his admission to hospital in the evening.

On examination, he was a tall, florid, well nourished subject. His abdomen was of the semi-obese type and slightly distended. There was slight rigidity and pain on pressure in the left iliac region. His vomitus was offensive and dark brown in colour, rapidly becoming fæcal in type. His temperature was normal and his pulse rate 100. Between the vomiting attacks he was fairly comfortable. A diagnosis was made of intestinal obstruction.

Operation was undertaken through a lower abdominal mid-line incision. The abdominal contents were explored and, apart from an area of congestion on the sigmoid colon, showed no pathological change. The congested area of the sigmoid was traced to the lateral border, where a tightly twisted *appendix epiploica* was found. This was shiny black and twisted so tightly at its junction with the colon that the peritoneal covering of the colon had become drawn tightly together in strands, like a web, to the extent of fixing the sigmoid in a partial kink. The whole area of the colon involved, even across to the mesocolon on the medial border, was covered with dilated veins and congested blood vessels.

The *appendix epiploica* was removed, the descending colon restored to its normal contour, and the patient made an uneventful recovery.

Dr. S. S. Gardiner kindly examined the abdomen with me at operation in order to check the findings.

Comment.

The two cases are interesting because of their dissimilarity and the fact that the clinical signs and symptoms varied according to the location of the *appendix epiploica* on the large intestine. They, moreover, illustrate the difficulty of classifying the symptoms and signs into a definite clinical syndrome. The second case is interesting because of the severity of the symptoms, and the first because the *appendix epiploica* involved was situated on the caecum, whereas the most common site is the sigmoid.

The cause of torsion is still obscure, but there are good grounds for believing that it takes place accidentally during the mass peristaltic movements of the colon, in which the contained bolus is forced through a considerable length of the large bowel.[2] These movements are becoming better understood following recent research at the Mayo Clinic, where motion picture films have been taken showing the normal intestinal movements in various laboratory animals.

It is reasonable to assume that, if the movements of an *appendix epiploica* which naturally accompany the long peristaltic waves of the colon to which it is fixed by a process of the peritoneum, were impeded in any way by adhesions or by disease, torsion would result. In furtherance of this theory one could surmise also that any sudden disturbance of the hydrostatics of the abdominal cavity (about which a number of theories have recently been propounded) would perhaps be sufficient to initiate twisting in an *appendix epiploica*; for example, the spasms of coughing mentioned in the history of Case I.

References.

(1) G. H. Colt: "Three Cases of Torsion of an Appendix Epiploica of the Sigmoid Colon", *The British Journal of Surgery*, Volume XIX, January, 1932, page 508.
(2) Editorial, *The British Medical Journal*, January 2, 1932, page 22.

ACUTE CARBON MONOXIDE POISONING IN A MOTOR CRUISER.

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I WAS recently called to a forty-foot motor cruiser to attend the whole party aboard in a state of collapse. The story was that some twenty minutes before the cruiser reached her mooring, one of the party complained of malaise and nausea. Within a few minutes two of the

others collapsed, shortly followed by the remainder on board, seven in all. With extreme difficulty they managed to get the ship moored and send for help. By the time I arrived on the scene three had more or less recovered, and four were still collapsed.

The symptoms were as follows: One and all experienced a prodromal and warning symptom—a thumping sensation in the head, especially in the occipital region. Several of the patients remembered no more till they more or less recovered, though they were never unconscious. Later symptoms were extreme weakness, collapse, retching (only the most severely affected patient had persistent vomiting), muscular twitchings and headache.

On examination, their colour, in spite of collapse, was notably good; the pulse was rapid and thready; the knee jerks were present and the pupils dilated but reacting.

Four of the patients recovered fairly rapidly, as the engine was stopped, the whole ship ventilated, and the patients removed into fresh air and given stimulants. Two took some hours to recover sufficiently to be taken home, while one was severely collapsed, with persistent vomiting. He was nursed on board that night and was well enough to go home next day. I consider he had a close call. All suffered from fairly severe headache for some thirty-six hours.

On investigating the cause, I found that the ship was a motor cruiser of about forty feet in length. She had a large forward sleeping cabin, a large rear enclosed well deck, and a raised centre section, totally enclosing the engine-room in a space about twelve feet by eight feet by four feet six inches, with no external ventilation whatsoever. On top of the engine housing was the wheelhouse, with sliding glass windows all round. The day was a wretched one, with a cold southerly blowing and intermittent, cold, drizzling rain, wherefore the crew had closed up almost all the portholes and windows. Furthermore, they were preparing the ship to be left at her mooring, which probably accounted for more closing up than the weather. There was no detectable odour whatever from the engine-room. I found that the engine had been running on an extremely rich mixture, with an excessive petrol consumption, accounting for the formation of carbon monoxide.

That there must have been a leak somewhere in the exhaust system was obvious, but structural difficulties prevented me investigating this at the time. The leak has since been found and eliminated, and through and through independent external ventilation of the engine-room is being installed on my recommendation.

The above-mentioned type of pleasure cruiser, with totally enclosed engine housing, is fairly common and must always be a potential source of danger.

Reviews.

PROGNOSIS.

THERAPEUTIC advances since the publication nine years ago of the third edition of "An Index of Prognosis and End-Results of Treatment",[1] by various writers under the editorship of Rendle Short, have altered the prognosis of many disorders. Insulin and liver therapy for *diabetes mellitus* and pernicious anæmia respectively exemplify this. The present edition has been thoroughly revised.

The authors had two main objects in view: first, to consider the end-results of different methods of treatment, with their advantages and disadvantages, and, secondly, to provide data, apart from any question of treatment, to assist in forming a rational prognosis in the individual patient. General practitioner and specialist alike will particularly appreciate the success attained with the first of these objects.

To determine end-results of treatment, it is necessary to use statistics which vary in accuracy according to their source. This factor receives consideration in the summing up of each discussion. Quoted figures form an unobtrusive part of the text, though frequently they could be further reduced by deleting older statistics which are only of historical interest. For instance, data relating to ligation of the thyreoid arteries in exophthalmic goître are unimportant, since preoperative iodine therapy has relegated this operation to the past. The use of iodine has so improved operative results in this disorder that reports of statistics dating back to the time of Kocher seem unnecessary.

Geographical conditions seldom appreciably alter the prognosis of individual disorders, so that in general the Continental, British and American statistics quoted may be taken to apply to this country. Reference to any Australian literature is rare, and this is particularly disappointing in the discussion on hydatid disease. It is stated that the principal methods of treatment of the condition are aspiration or enucleation of the cyst. The risks attending aspiration are reviewed, but this measure does not meet with the unqualified disapproval it deserves.

In a volume of this type it is easy to find subjects for criticism. For example, it is stated that in exophthalmic goître, unless the elaborate method of anoci-association of Crile is employed, the choice of anæsthetic is limited to ether or chloroform. A subtotal thyreoidectomy can practically always be done under nitrous-oxide oxygen anæsthesia preceded by the injection of morphine and either atropine or hyoscine. Chloroform should never be used for a patient with exophthalmic goître.

In the surgical treatment of this condition it is stated that normally a hemithyreoidectomy may be performed and some months later the subtotal removal of the gland may be completed. In competent hands today a one-stage subtotal thyreoidectomy can be successfully performed in the vast majority of instances. The quotation that a high preoperative glycosuria resisting medical treatment contraindicates operation is out of date since the introduction of insulin. This is tacitly admitted later. The patient with diabetes complicated by thyreotoxicosis is almost always benefited by a subtotal thyreoidectomy.

Despite any such criticisms, this volume can be recommended as a most useful addition to the library of the practitioner, for it is one to which constant reference, seldom, if ever, without benefit, will be made.

[1] "An Index of Prognosis and End-Results of Treatment", by Various Writers; Edited by A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S.; Fourth Edition; 1932. Bristol: John Wright and Sons Limited. Imperial 8vo., pp. 610. Price: 2 guineas net.

HAY FEVER.

WILLIAM LLOYD has republished his little book on hay fever and hay fever asthma.[1] He gives an excellent short historical account of researches into its cause and treatment. The pollen of grasses is regarded as the cause of true seasonal hay fever, which appears to be much more prevalent in America than in Great Britain. The work of Blackley, Dunbar and Wyman is clearly described. The author claims that the attacks can be largely prevented or markedly lessened by carrying out prophylactic treatment to exclude pollen from the dwelling rooms, and by wearing glasses and a porous rubber apparatus over the nose. Nasal douching and nasal sprays are recommended, and treatment of nasal defects, especially by cauterizing sensitive areas on the nasal mucosa. Oil of chamomile is used as a nasal spray. He has not found pollen vaccines or antitoxins effective. Mention is made of the use of hot strong coffee on an empty stomach. Adrenalin, ephedrine, cocaine and menthol are among the medicaments suggested for application to the nasal mucosa. The prophylactic methods are rather elaborate and perhaps difficult for the average patient to carry out. Great stress is laid on the treatment of the nose by surgical methods. Illustrative cases are given at the end of the book; the results reported are certainly very satisfactory.

[1] "Hay-Fever, Hay-Asthma, Its Causes, Diagnosis, and Treatment", by W. Lloyd, F.R.C.S.; Third Edition; 1931. London: Straker Brothers. Demy 8vo., pp. 130, with illustrations.

The Medical Journal of Australia

SATURDAY, MAY 28, 1932.

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LYMPHANGITIS AND CANCER.

THE work of Mr. Sampson Handley on the dissemination of cancer by the lymphatic system is known in every part of the scientific world and is accepted by the majority of investigators. Whether surgeons know it or not, they have accepted his work, for they have fashioned the steps and the extent of many of their operations to conform to his views. For upwards of twenty years Mr. Handley has been pursuing his studies; he has become more and more impressed with the importance of the rôle played by the lymphatic system in the origin of cancer and has published the results of his researches in an important book, "The Genesis of Cancer".[1] Mr. Handley's work has always commanded such respect and his sincerity is so obvious that an attempt should be made by all those interested in the problem of malignant disease to understand his teaching. The statement is sometimes made that the study of morbid anatomy has nothing further of importance to offer to the investigator, that nothing further can be learned by observation of structural changes in the cell. Physicists, biological chemists, radio-therapy workers and the like occupy the centre of the stage at the moment, the limelight (such as it is) is centred around them, and hope for the future would appear to depend on the success of their efforts. They are not so foolish as to ignore the solid foundation of morbid anatomy by which much of their work has been made possible. If Mr. Handley's views can be substantiated, physicists and chemists may find the field of their investigations more restricted, and the day when the loose ends will be woven into a completed strand will be brought nearer.

Mr. Handley's fundamental claim is that chronic lymph stasis is a constant factor in the pathogenesis of cancer. He admits "certain imperfections" in the evidence, but holds that "the degree of proof is as high as can reasonably be demanded at the present time in such a complex and difficult subject". Starting with a consideration of lymphangitis, he shows that the effect of lymph stasis in the skin is to cause active proliferation of the connective tissue cells and of the lymphatic endothelium in the affected district. The fibrous tissue swells and hypertrophies, and on the surface of the skin papillomata are produced. The epithelium covering the enlarged papillæ then undergoes hypertrophy. Chronic lymphangitis is brought about by irritants, whether they be bacterial, physical or chemical. Mr. Handley devotes a great deal of attention to lupus and claims to have demonstrated the lymphangitis-papilloma-carcinoma sequence in lupus carcinoma. He also claims that there is much evidence bearing on the genetic relationship of papilloma or adenoma and carcinoma. In reply to critics of a previous communication, he admits that it would be incorrect to say that papillomatosis precedes every variety of carcinoma, unless adenomatosis is included under the term papillomatosis. He points out that lymph stasis may occur without giving rise to either papillomatosis or adenomatosis, and that even so his lymph stasis theory is not invalidated.

This short statement will give some indication of Mr. Handley's views. It would be easy to pick holes in his argument, and adverse criticism of his views

[1] "The Genesis of Cancer", by W. S. Handley, M.S., F.R.C.S.; 1931. London: Kegan Paul, Trench, Trubner and Company, Limited; Australia: Angus and Robertson Ltd. Demy 8vo., pp. 274, with illustrations.

has already appeared. He, however, admits to imperfections. If it can be proved that lymph stasis is always a precursor of cancerous change, it becomes necessary to discover what are the additional factors operating in the affected area—cancer does not always arise in areas of lymph stasis. The additional factors will probably be chemical or physical; they may be the result of lymph stasis *per se* or of some independent quality. This is where the physicist and chemist will continue their researches.

This article is not intended as a comprehensive review of Mr. Handley's book; it will be reviewed by each and every medical practitioner concerned with cancer in ward or laboratory. The clinician will be impressed with the value of accurate observation, and the experimental pathologist should, as Mr. Handley suggests, find a stimulus and attain a balance necessary to his own work. It was unnecessary for Mr. Handley to point out in his preface that the book is not an "armchair speculative essay".

Current Comment.

RHEUMATOID ARTHRITIS AND A CAUSATIVE STREPTOCOCCUS.

J. W. GRAY AND C. H. GOWEN give an account of investigations concerning rheumatoid arthritis (*arthritis deformans*).[1] They state that an arthritic tendency appears to run in some families and that certain types of individual are prone to arthritis. Adjuvant factors are trauma (which induces lowered resistance in a joint) and fatigue, especially if associated with cold and wet. Focal infection and bacteriæmia may exist without arthritis, but if resistance be lowered by exposure or fatigue, conditions become favourable for localization of infection in the joints. Possibly a climatic factor may exist. The prevalence of upper respiratory infections in some climates may account for joint infections. Dry, equable climates are ideal for "rheumatic" patients. The relation of focal infection to arthritis was noted twenty years ago. E. C. Rosenow isolated *Streptococcus viridans* in 1924 from lymph glands in cases of chronic infectious arthritis. Most investigators regard streptococci as the bacterial agents responsible for this disease; but others think that staphylococci, gonococci, diphtheroid bacilli and other organisms may also produce a deforming arthritis. R. L. Cecil, E. E. Nichols and W. J. Stainsby in 1929 reported that in 154 cases of chronic infectious arthritis 62·3% of blood cultures yielded a short-chained streptococcus. The same organism was yielded by 33 out of 49 joint cultures. Control blood and joint cultures were all sterile. Using a special technique, these investigators produced typical deforming arthritis in rabbits' joints by inoculation with the "typical strain" of streptococcus from a patient's blood, and the organism recovered from rabbits' blood was found by cross-agglutination to be the same as the original organism.

Gray and Gowen used an improved method for quick growth of the organism and reported that the streptococcus cultured from the blood and joints was similar to the one deemed by Cecil to be specific for rheumatoid arthritis. It was a Gram-positive coccus occurring in chains of two to twenty. It was bile-insoluble and did not ferment inulin. On streaked blood agar plates there was a delicate growth of greyish isolated colonies showing production of methæmoglobin of a pale green shade. After twenty-four to forty-eight hours' incubation there was a narrow zone partly hæmolyzed around the colony. Having intermediary qualities, the organism has been termed α prime streptococcus. Typical α or *Streptococcus viridans*, found in rheumatic fever, produces a much wider green zone without hæmolysis. Alpha type streptococci have been recovered from joint fluid, joint tissues and from primary foci. Cecil termed the intermediary type the "typical" strain for rheumatoid arthritis. In the *arthritis deformans* group, of 71 patients, 62% harboured this organism in the blood or joint fluid or both. Of thirteen rheumatic fever patients five harboured the organism. Three patients (sufferers from malignant endocarditis, acute peritonsillar abscesses and subacute osteitis) yielded the α type. Of 71 control persons with osteoarthritis, normal persons and sufferers from miscellaneous diseases, only two manifested β streptococcus. Of the 71 patients with *arthritis deformans*, 56 had tonsillar, tooth or sinus infections (or a combination of such lesions). One infection declared itself after acute appendicitis, one was associated with gall-bladder infection and two with peptic ulcers.

The clinical course of *arthritis deformans* is characteristic of an infection. The onset may be acute, febrile and be accompanied by acute migratory joint symptoms suggestive of rheumatic fever. When the onset is gradual, there is progressive insidious swelling of the joints. Weight and strength are lost. Metastatic infection may be suggested. Two of the patients of Gray and Gowen had iritis before the joint symptoms appeared. Pathologically the joint lesions are typical of an infectious inflammatory reaction. Bacteriological differentiation from rheumatic fever may be difficult. In primary cultures there is usually a slight difference in the characters of the streptococci found in the blood and joints of these two entities. Agglutination tests may be of value. Possibly rheumatoid arthritis may develop by specific infection after rheumatic fever.

[1] *The American Journal of the Medical Sciences*, November, 1931.

Diagnosis of rheumatoid arthritis from osteoarthritis also may be difficult. In osteoarthritis there is no history of focal infection and there are no pathological features of infection. But there are mixed cases only distinguishable bacteriologically. Cecil found that patients with rheumatoid arthritis showed high agglutination for the "typical" strain of streptococcus in 94% of the cases. In Gray and Gowen's series, in 52 of 60 cases of rheumatoid arthritis, agglutination of the rheumatoid streptococcus occurred. Most of the cases of *arthritis deformans* of long duration manifested agglutination in a low dilution, if at all. The agglutination reaction apparently indicates that an individual is developing a resistance to an infection somewhere in the body. The streptococcal agglutination test will probably not be so specific as the Widal reaction, since the typhoid organism is in a narrow group and the streptococci are heterogeneous. The agglutination reaction is valuable in diagnosis, especially from osteoarthritis. In treatment it was not possible to develop an immune serum. A specific vaccine was prepared from a type streptococcus isolated from the patient's blood, joint fluid or septic foci or, if autogenous vaccine were not procurable, from typical strains of the organism. Vaccine made from blood or joint fluid is more specific than from other sources, because different strains may vary greatly. In early and moderately advanced cases specific vaccine gave very good results, and when it was given before removal of septic foci it was found that it might prevent undesirable joint reactions. Intravenously administered vaccine gave rapid improvement. Such quick response may be due to desensitization of hyperergic tissues. The dose and intervals between injections must be regulated to obviate reactions, especially in the joints.

The investigations of Gray and Gowen are not conclusive, and it is difficult at times in their communication to keep on the track of their particular organism. A few years ago N. Mutch isolated *Bacillus fallax*, an intestinal anaerobe, from cases of chronic arthritis and obtained good results from a corresponding vaccine. Hæmolytic streptococci of different strains have been isolated in cases of acute and subacute periarthritis and exudative periarthritis. The joints in chronic arthritis, not tuberculous or syphilitic, have yielded staphylococci and other organisms, and the lymph glands gonococci and *Streptococcus viridans*. The therapeutic test of vaccine is not conclusive. Good results have been recorded in rheumatoid arthritis from non-specific vaccines or other form of protein shock therapy. The nomenclature of chronic arthritic conditions is bewildering. With some authors there are three types: infective, atrophic and hypertrophic. Others include atrophic and infective forms with rheumatoid arthritis. Spondylitis is apparently osteoarthritis. The term *arthritis deformans*, which once included osteoarthritis and rheumatoid arthritis, should be abolished. Where should Still's disease be placed? Progress is impossible until escape is made from the babel of terms. Even if a prime streptococcus be constantly and exclusively present in rheumatoid arthritis, does inquiry into the ætiology end with the discovery of the organism? Septic foci are only an episode in the disorder. Constitutional diathesis must be considered. Perverted metabolism and deranged endocrine action are important. In many forms of chronic arthritis an abnormal metabolic rate has been found, and the arthritic condition and the metabolic rate have improved on thyreoid administration. What is the significance of hypochlorhydria in rheumatoid arthritis? Rheumatoid arthritis and osteoarthritis frequently coexist. In rheumatoid arthritis may occur subcutaneous nodules indistinguishable from those of orthodox rheumatism. The different forms of chronic arthritis cannot be definitely classified as distinct entities.

CYSTITIS EMPHYSEMATOSA.

CYSTITIS EMPHYSEMATOSA receives scant mention in the literature. Apparently it is a very rare disease. In fact, it has not been generally recognized as a clinical entity. It is characterized pathologically by the presence of gas-filled vesicles in the bladder wall, by swelling of the mucosa and submucosa, and by areas of hæmorrhage between the vesicles. It is a disease of adults, though in one of the twenty-four reported cases the patient was a child of twelve years. Women appear to be more liable than men. Schönberg isolated from the bladder wall a gas-forming organism possessing the general characteristics of *Bacillus coli*. Laboratory animals infected with this organism developed a condition similar to *cystitis emphysematosa* of man.

Abraham Ravich and Perry Katzen recently reported a case of this disease.[1] They believe they are the first to diagnose the condition during life. Their patient was a man who suffered from severe hæmaturia, frequency of micturition and dysuria. Through a suprapubic cystostomy wound the mucosa of the bladder was observed to be intensely inflamed and œdematous, and marked with innumerable clear vesicles that varied in size from a pin's head to a pea; there was generalized oozing of blood from the mucosa. The inflammation rapidly subsided as a result of suprapubic drainage. Later a large retention diverticulum was removed surgically and the patient recovered. *Bacillus coli* was isolated from the urine. Ravich and Katzen believe that their patient's life was saved by means of prompt suprapubic drainage. It is worthy of note that a patient may have no obvious symptoms of urinary disease. This fact is rather suggestive of the possibility that the pathological appearances ascribed to *cystitis emphysematosa* may be due to *post mortem* changes. The case reported by Ravich and Katzen seems to disprove this view. Possibly, as these observers suggest, the disease may be much commoner than the numbers of reported cases would indicate.

[1] *The Journal of the American Medical Association*, April 9, 1932.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Lipoid Nephrosis.

L. BERNARD AND J. PARAF (*La Presse Médicale*, October 31, 1931) discuss tuberculous hydræmic nephritis and lipoid nephrosis. Lipoid nephrosis, distinguished by the presence of refractile bodies in the urine and hyperlipæmia, has rarely been observed in France. Lipoid nephrosis is merely hydræmic nephritis in which these urinary and humoral peculiarities are noted. Muller, Volhard and Faber in 1905 described lipoid nephrosis and emphasized the good prognosis in that condition. French observers have not found the result of high protein diet and thyreoid treatment so satisfactory in relieving the symptoms in this type of nephritis. Three cases are quoted in which hydræmic nephritis with lipuria, refractile bodies in the urine and hyperlipæmia occurred; in all three cases pulmonary tuberculosis preceded or accompanied the renal abnormality, and treatment with full diet and by other measures used in tuberculosis was indicated. These observations and similar records by other observers indicate the frequency with which tuberculosis accompanies so-called lipoid nephrosis, and it is suggested that a careful search for an active tuberculous focus should be made in all cases of supposed lipoid nephrosis.

Experimental Focal Infection and Cardiac Structure.

N. W. JONES AND S. J. NEWSOM (*Archives of Pathology*, March, 1932) have investigated the effect of experimentally produced focal infection on the hearts of dogs. The pulp was removed from the canine teeth of the dogs and the pulp cavity was filled with a culture of streptococci, dental cement was placed in the cavity, and the tooth was permanently filled with silver amalgam. The dogs, with some control animals, were then subjected to stress and strain by daily exercise to exhaustion. At the end of the experimental period the dogs were killed by asphyxia during deep ether anæsthesia. Dental abscesses were found in all inoculated dogs. There were no other constant extracardiac structural changes. The hearts of inoculated dogs constantly showed very small vegetative or verrucose mitral and/or aortic endocarditic lesions, patchy parenchymatous degeneration, nuclear changes, increase in the diameter of the muscle cell, and a slight round cell infiltration. A positive relationship between the dental infection and cardiac hypertrophy as measured by diameters of muscle cells was noted. The authors hold that direct measurement of diameters of cardiac muscle fibres, under their experimental conditions, is a more reliable criterion of cardiac hypertrophy than ratios of heart weight to body weight or of heart weight to body surface area. Strain and stress in the absence of focal infection did not affect the gross or the microscopical characteristics of the heart.

The Spleen in Streptococcal Infections.

J. W. ORR (*The Journal of Pathology and Bacteriology*, March, 1932) points out that acute enlargement of the spleen almost invariably occurs in septicæmic conditions, especially those due to streptococci. Since it is clear that some part in this enlargement is played by the splenic tissues themselves, apart from invasion by hæmic leucocytes, the author has conducted experiments in an attempt to follow out the sequence of changes. He used various strains of hæmolytic streptococci and made subcutaneous injections into the lumbar region of rats. Complete examination of the spleen was carried out by means of various fixing methods and stains. The two observations to which the author attaches most importance are, first, that the peri-Malpighian region plays an important rôle in the reaction of the spleen to streptococcal infection, and, second, that the earliest response is made by the lymphoid and reticulo-endothelial cells of the spleen itself, polymorphonuclear cells becoming active only at a somewhat later stage. The greatest primary activity is found in the peri-Malpighian zone, at the boundary between the lymphoid follicles and the pulp. Proliferation of reticulum cells with disappearance of lymphocytes is followed by marginal accumulation of neutrophile leucocytes. Subsequent changes in the pulp are congestion, infiltration with polymorphonuclear cells and multiplication of reticulum cells and later of lymphocytes and plasma cells. Progressive decrease in the size of Malpighian bodies occurs. The author discusses the reduction in the number of lymphocytes. The reticulum cells display their greatest activity when they are associated with seemingly inert lymphocytes. Some interaction takes place between the lymphocytes and reticulum cells in the peri-Malpighian zone. The author suggests that lymphocytes may, under the influence of an infective stimulus, be transformed into macrophages.

Healed Dissecting Aneurysm.

THE term "healed" has been applied to dissecting aneurysms which communicate with the lumen of the aorta at or near their origin and termination, and which become lined with well-formed fibrous connective tissue covered by endothelium, the canal thus assuming the characters of a blood vessel and taking on part of the functions of the main channel. T. Shennan (*The Journal of Pathology and Bacteriology*, March, 1932) has described a case which he claims to be unique. The aneurysm occurred in a male patient, aged sixty-four years. In this instance there was a complete healing up of a dissection along the outer layers of the *tunica media*; no channel persisted, the effused blood having been completely organized and replaced by a thick layer of vascular connective tissue containing numerous cells full of hæmatogenous pigment. Shortly before death two new dissections developed; the second caused death by rupturing into the pericardial sac.

Parathyreoid Tumours without Osteitis Fibrosa.

G. HADFIELD AND H. ROGERS (*The Journal of Pathology and Bacteriology*, March, 1932) report two tumours of the parathyreoid gland. One occurred in a woman, aged fifty-eight years; the other in a man, aged fifty-one. The bulk of the tumours was "relatively enormous". In one there appeared at first to be some reason for regarding the enlargement as being ætiologically related to the skeletal changes of frank acromegaly, associated with a large chromophil adenoma of the pituitary. In the other an adenoma, indistinguishable as far as could be judged by ordinary histological methods from those described in generalized *osteitis fibrosa*, was associated with a radiologically normal skeleton. The authors have been able to find only six previously reported cases of parathyreoid adenoma or hyperplasia associated with pituitary disease. They do not think that this association is so rare as this figure indicates. They think that the skeletal overgrowth of acromegaly alone should, theoretically, make excessive demands on the parathyreoids. After some argument they conclude that in the first of their two cases the adenomatous enlargement of the parathyreoid was unrelated to the skeletal changes; in other words, they place it in the same category as their second case. They point out that since Mandl restored the calcium metabolism to normal in a case of *osteitis fibrosa* by excision of a parathyreoid tumour, and thus disproved Erdheim's theory according to which the parathyreoid adenomata in these cases were secondary to the disease of the skeleton, the publication of similar cases has given rise to the impression that a parathyreoid adenoma must of necessity be associated with diffuse skeletal softening. They hold that their two cases prove this to be fallacious. In their opinion it seems likely that a parathyreoid tumour, closely resembling the normal gland in structure, may occur without elaborating any excess of internal secretion.

Renal Lesions in the Toxæmias of Pregnancy.

E. T. BELL (*The American Journal of Pathology*, January, 1932) divides the toxæmias of pregnancy into: (i) typical eclampsia with convulsions, (ii) eclampsia without convulsions, (iii) preeclampsia, (iv) *hyperemesis gravidarum*, (v) pregnancy in association with preexisting renal disease. He has studied the kidneys in twenty cases of toxæmia of pregnancy. He concludes that in fatal cases of

eclampsia a characteristic glomerular lesion is found. The glomeruli show a definite narrowing of all their capillaries, caused usually by an increase in thickness of the capillary basement membrane, but sometimes by an increase of endothelial cells. The author reports one case in which the lesions resulted from an attack of eclampsia which occurred seven years before death. These consisted of focal hyaline areas in the glomeruli with partial or complete glomerular obliteration and varying degrees of tubular atrophy. A peculiar form of chronic renal disease may result from the eclamptic kidney. In one case of *hyperemesis gravidarum* glomerular lesions were found which corresponded to those of typical eclampsia. In three other cases the glomeruli were normal. A fatty liver without necrosis is characteristic of *hyperemesis gravidarum*. When a woman with chronic renal disease becomes pregnant, an aggravation of all the nephritic symptoms usually occurs. The condition cannot be distinguished from pre-eclampsia and eclampsia unless the condition of the kidneys prior to pregnancy is known, or unless there is a definite impairment of renal function. Chronic nephritics show no special tendency to develop eclampsia.

MORPHOLOGY.

The Intraglandular Cleft of the Pituitary Body.

JOHN BRANDER (*Journal of Anatomy*, January, 1932) states that between the capsule of the pituitary gland and the periosteum lining the *sella turcica*, or alternatively, between two layers of the *dura mater* of this region, there is a relatively large blood space which almost completely envelops the organ and communicates at several points with the blood supply of both anterior and posterior lobes above, and with the marrow of the sphenoid bone below. He states also that it appears that the lower end of the intraglandular cleft remains patent and in connexion with the blood space just referred to, thus leaving a possible course to be followed by the colloid material within the cleft when passing to the general circulation.

The Function of Intraarticular Fibro-Cartilage.

M. A. MACCONAILL (*Journal of Anatomy*, January, 1932) states that it is shown by reference to the theory of lubrication that intraarticular fibrocartilages are to be related primarily to the synovial fluid rather than to the articular surfaces of the bones. They act to bring about the formation of wedge-shaped films of synovia in relation to the weight-transmitting parts of joints in movement. These wedges are narrowed in the direction of motion, and are necessary for weight transmission. They are to be found in joints where thrusts are likely to bring about a premature approximation of the joint surfaces. Preparations are described of knee and inferior radio-ulnar joints, that demonstrate the action of such cartilages. These also show that the cartilages are congruent with articular surfaces only in the weight-carrying position of the joints.

Osteogenic Capacity in Vitro.

H. B. FELL (*Journal of Anatomy*, January, 1932) states that periosteum removed from embryonic limb bone rudiments of six and ten days' development is able to ossify *in vitro*, although by the conditions of the experiment it is deprived of its normal association with cartilage and of a blood and nerve supply. Periosteum from late embryos and young chicks may grow profusely *in vitro*, but only ossifies in rare cases, probably owing to severe damage inflicted mechanically on the osteoblastic layer when the tough, firmly attached fibrous membrane is being stripped away. Undamaged endosteal osteoblasts derived from the Haversian spaces of late embryonic and early post-embryonic tibial bone readily form bone *in vitro*.

The Tracheo-Bronchial Lymphatic Glands.

H. P. NELSON (*Journal of Anatomy*, January, 1932) describes in detail the glands found in the mediastinum and hila of the lungs, together with a brief reference to the other visceral glands in the thorax. A new and simple terminology for the bronchial glands is presented and a terminology for the bronchi is presented that is based on their area of distribution rather than on the morphology of the lung.

Fossil Brains of Early Tertiary Mammals.

F. TILNEY (*Bulletin of the Neurological Institute of New York*, November, 1931) gives an account of an investigation of fossil mammalian brains from the tertiary period in North America. He states that the tertiary mammalian brains present distinctive marks of the reptile which are unmistakable and exhibit certain critical stages through which the evolutionary process passed from the beginning to the middle of the age of mammals.

The Sensory Component of the Spinal Accessory Nerve.

W. F. WINDLE (*Journal of Comparative Neurology*, August, 1931) finds that, contrary to usually accepted statements, the spinal portion of the eleventh cranial nerve in the monkey and cat is a mixed nerve. Some of its sensory components have been located in the nerve itself and others in the first cervical spinal ganglion of the cat.

The Follicular Cells of the Thyreoid Gland.

J. F. NONIDEZ (*American Journal of Anatomy*, January, 1932) records the occurrence in the thyreoid glands of puppies, from birth to six months, of large epithelial cells, which arise in the follicular epithelium and later migrate into the inter-follicular spaces, remaining close to the follicles. The cells contain argentophile granules, which, the writer states, probably represent the antecedent of an endocrine secretion. The fate of these cells is unknown, but the writer believes that the segregation of parafollicular cells during early post-natal life is evidence of the presence of two types of elements with different potentialities in the follicular epithelium.

The Development of the Ciliary Ganglion.

E. DEERY (*Bulletin of the Neurological Institute of New York*, November, 1931) finds that both the oculomotor and the ophthalmic pathways contribute cells from the neuraxis to the formation of the ciliary ganglion. The migration along the oculo-motor pathway is relatively small; but such cells are probably the first to form the ciliary anlage.

The Atrio-Ventricular Conduction System.

J. CARDWELL AND D. ABRAMSON (*American Journal of Anatomy*, November, 1931) furnish a report concerned with the macroscopical distribution of the conducting system in the ventricles of the heart of the ox, as revealed by multiple injections and subsequent dissection of more than fifty specimens. Their observations confirming and extending those of previous workers clearly indicate that the bundle of His, its right and left branches, and the sub-endocardial and myocardial Pürkinje network, constitute a single conducting system.

The Innervation of the Superficial Facial Musculature.

W. K. SMITH (*The Anatomical Record*, October, 1931) states that a combined morphological and experimental study of the motor innervation of the superficial facial and superficial cervical muscles in the opossum brought forth conclusive evidence that this field is supplied exclusively by the facial nerve. These muscles therefore represent a phylogenetically uniform muscle group innervated exclusively by the facial nerve and so furnish additional evidence in support of the concept of a constant relationship between a muscle and its motor nerve, as set forth in the Fürbringer hypothesis.

The Cervical Sympathetic Connexions.

S. A. SIWE (*American Journal of Anatomy*, July, 1931) gives an account of dissections on both sides of several human fœtuses and of twenty adult human cadavers, with the special object of examining the cervical ganglionated cord and its connexions with the spinal nerves, with special attention to the *nervus vertebralis*. He also gives a few notes on the connexions between the cervical sympathetic system and the local cerebral nerves, particularly the vagus and its branches. He comes to conclusions which vary considerably from those of former investigators.

Medical Societies.

THE COUNCIL FOR MENTAL HYGIENE FOR NEW SOUTH WALES.

THE inaugural meeting of the Council for Mental Hygiene for New South Wales was held at the British Medical Association House, Macquarie Street, Sydney, on April 13, 1932, PROFESSOR HARVEY SUTTON in the chair.

Mental Hygiene.

PROFESSOR W. S. DAWSON introduced the subject of mental hygiene, and outlined a general survey of mental hygiene movements during the past century, making mention of the progress made in the United States of America, where, largely through the efforts of Mr. Clifford Beers, author of "The Mind that Found Itself", and at one time a mental patient, the National Council for Mental Hygiene had been founded in 1909. This Council had grown to large proportions, and had been well to the front in the Great War. Similar councils had been established in all leading countries.

Professor Dawson said that the matter of establishing a council in New South Wales had been considered during the past three or four years, and some of those present at the inaugural meeting had been present at a meeting held in 1929 for the purpose of discussing the subject. These representatives had also attended a discussion on mental hygiene at a sectional meeting of the Australasian Medical Congress, held in Sydney later in the same year, at which it had been urged that the establishment of a Council was highly desirable. Towards the end of last year the Section of Neurology and Psychiatry had moved that something further be done, three or four members had collaborated, and the meeting that evening was largely the outcome of their deliberations.

Professor Dawson said that a good deal was known of the preservation of physical health. Mental hygiene was aimed at the achievement of somewhat similar objects, in improving the well-being of the community, by insuring that mental processes were working at their fullest efficiency and by preventing and treating mental disorders of various kinds. Mental conditions in adults, he said, were in many cases the culmination of maladjustments in childhood and the home setting *et cetera*. There was therefore need of provision for the early treatment of mental and nervous disorders. He paid tribute to the excellent psychiatric clinic at Broughton Hall, which, he said, was the envy of Victoria and other States. There was considerable reason for pleasure at what had already been attempted in this State, but there was always room for further development. The Council for Mental Hygiene should form the meeting ground for experts, social workers and others, who came together to help one another in the problems of delinquency, criminality and social maladjustments of various kinds. There was a great need for child guidance clinics, for the investigation and treatment of "problem" and difficult children, and for those who were below the average in mentality, yet physically up to the average. Some thought it was a bad time to start such a movement, but there was a great deal to be done before funds could be spent to good advantage, and in that way the Council could prepare for better times. Much could be done in the way of making surveys, collecting statistics *et cetera*. This work would merely cost the time and experience of those willing to work.

DR. A. W. CAMPBELL said he approved, without hesitation, of the objects outlined in the circular. He said there was a great need for psychiatric wards and out-patient departments in general hospitals, for the treatment of patients with minor psychoses, also for child guidance clinics. The ever-increasing army of epileptics made him long for the day when the colony or village system would be developed in this country, so that the sufferers could be made more useful and contented. Then there was the question of the sterilization of the mentally unfit, which was exercising the minds of many. Dr. Campbell said he wished the Council every success.

MRS. MUSCIO, on behalf of the National Council of Women, said she was very pleased to learn of the revival of the mental hygiene movement, and looked forward to its help and guidance in the social work of the National Council of Women. She and her Council were willing to help in any way possible.

DR. A. H. MARTIN, representing the University Department of Psychology, in the absence of Professor Lovell, and the Institute of Industrial Psychology, said he had been one of those present at the meeting held in 1929; he assured the meeting of the whole-hearted support of the departments he represented. He discussed the relation of mental hygiene to the proper adjustment in industry of the adolescent.

JUDGE BEVAN, on behalf of the Racial Hygiene Association, said it was a well known fact that a very large percentage of the younger prisoners in the gaols, if not mentally deficient, were suffering from a retarded development and, but for that, in all probability, they would not be there at all. Mental hygiene could go a long way in preventing criminality, delinquency *et cetera*, and it was in the country's interest to do something to treat, rather than punish, a number of men who were detained in the gaols. He advocated keeping a record of the mentality of every boy or girl who passed through private or public schools, as a help towards preventing maladjustments *et cetera*.

PROFESSOR HARVEY SUTTON said that the interest in the community, amongst trained people in particular, in regard to mental hygiene had been very greatly improved in recent years. He made mention of the excellent work being done at the Children's Court, started by Dr. Davidson, now carried on by Dr. Bruce. More should be done to remove the stigma so frequently attached to mental illness. The whole problem was one viewed with a great deal of suspicion; thus the institution of early treatment was hindered. Professor Sutton said he had found it impossible to find text books on mental hygiene for ordinary intelligent people. There was a great need for such a council as this in the community. He concluded with the quotation: "Soundness of mind is the master key of human progress."

It was resolved that The Council for Mental Hygiene for New South Wales be established, and those present resolve themselves into a general committee, with power to add to their number.

Election of Officers.

The following office-bearers were duly elected:

President: Professor Harvey Sutton.
Honorary Treasurer: L. W. Phillips, Esq.
Honorary Secretary: Professor W. S. Dawson.
Executive Committee: The above-named office-bearers and Dr. Evan Jones, Dr. J. A. L. Wallace, Dr. H. M. North, Mr. Ebsworth, Mr. G. R. Thompson.

Constitution and Business.

It was resolved that the Executive Committee should draw up a constitution and report at a general meeting to be held at some date towards the end of May, 1932.

It was resolved that the Executive Committee be given authority, before the general meeting, to deal with the general business of the Council and, if necessary, to appoint subcommittees.

The meeting closed with a vote of thanks to the Chairman and to Professor Dawson for all he had done in preparing for the inaugural meeting.

THE MELBOURNE PÆDIATRIC SOCIETY.

A MEETING OF THE MELBOURNE PÆDIATRIC SOCIETY was held at the Children's Hospital, Carlton, on April 13, 1932, DR. REGINALD WEBSTER, the President, in the chair. The meeting took the form of a series of clinical demonstrations.

Diaphyseal Aclasis.

DR. J. G. WHITAKER showed a boy, aged four years, who was suffering from Keith's diaphyseal aclasis. There was

a progressive bowing of the right forearm. Examination revealed an apparent shortening of the right ulna with apparent overgrowth and bowing of the radius. A lump was also present on the vertebral border of the scapula. Dr. Macdonald's X ray report was as follows: "Imperfect development of the lower end of the ulna with osteochondromata and bowing, which suggest that the condition is an early expression of diaphyseal aclasis." Subsequent X ray examination showed the development of osteochondromata in various places, particularly the upper end of the left humerus. Many children on the mother's side of the family had been affected with "lumps on the bones". The blood did not react to the Wassermann test. Dr. Whitaker remarked that the disease had a marked disposition to run in families. The condition occurred principally at the growing ends of long bones and did not occur in membrane bones or bones composed of cartilage, such as the tarsus and carpus. It occurred only during the growing period. The deformity of the forearm, the arrest of growth of the lower end of the ulna, and the subluxation of the radial head were fairly constant.

DR. COLIN MACDONALD described the X ray appearances of diaphyseal aclasis, synonyms for which were "hereditary deforming dyschondroplasia" and "multiple osteochondromata". There were deformities in the diaphyseal extremities of the long bones in general, and there occurred multiple osteomata of the sessile variety and a characteristic deformity of the forearm, as seen in Dr. Whitaker's case, though this deformity was not always seen. The metaphyses of the long bones were broadened and irregular in outline, and the lamellæ were irregularly spaced without loss of lamellar structure. Diaphyseal aclasis might be contrasted with Ollier's disease, a chondromatous dystrophy affecting the extremities and shafts of the long bones. In the chondromatous areas there was loss of lamellar structure and there were no projecting osteomata. In reply to a question of Dr. Douglas Stephens as to the theories of the causation of diaphyseal aclasis, Dr. Macdonald said that although he had not heard of the theory that the sympathetic nervous system was at fault, the Scandinavian, Bentzon, believed that such was the case in Ollier's disease, and Dr. Roy Sear, of Sydney, had advanced the view that the very curious condition called melorheostosis (Leri) was in the same category.

Retarded Development.

DR. STEWART FERGUSON showed a male child to whom thyreoid extract had been administered. He had been admitted to hospital at the age of one year and eight months. The family history was irrelevant. The child had apparently been healthy until five months prior to admission, when he had begun to lose appetite and weight. He had become irritable, but did not appear to have pain. He had continued to walk till five weeks prior to admission, but from then on he had become increasingly feeble and had continued to lose weight. Occasionally he had seemed brighter, but altogether he had become steadily worse. There had been no diarrhœa or vomiting until two weeks before admission. His diet had consisted solely of cow's milk till the age of one year and three months, but from then on he had been given bread and milk, milk puddings and custards. Examination had revealed an extremely wasted, old looking child. The fontanelle was open and depressed, the muscles hypotonic, and the subcutaneous tissues dry and wasted. No other abnormality had been found. The weight at the time of admission was about 6·3 kilograms (fourteen pounds eight ounces).

During the seven months following admission numerous lines of investigation were carried out in an endeavour to find the cause of his failure to thrive. The blood did not react to the Wassermann test, even after a provocative dose of "Novarsenobillon". There was no reaction to the von Pirquet test. X ray examination of the radial epiphyses revealed no abnormality. The blood urea content was 28 milligrammes per hundred cubic centimetres. Blood sugar curves were normal, and analysis of the wet fæces revealed: total fat, 5%; fatty acids, 3·32%; and neutral fat, 1·68%.

He was fed on boiled skimmed milk, then boiled skimmed lactic acid milk, then boiled whole cow's milk and light baby diet, but without any sustained improvement.

Ultra-violet radiation, "Ostelin", "Radiostoleum", "Vita B", "Marmite", antitoxic serum, blood transfusion, glucose (administered intravenously), insulin therapy and mercurial inunctions all proved valueless, and his weight after seven months' treatment was only 7·2 kilograms (sixteen pounds seven ounces). A trial was then made with thyreoid extract in a dose of 0·0054 gramme (one-twelfth of a grain) twice daily. Improvement was almost immediate. The weight continued to rise steadily until, at the time of the meeting, seven weeks later, he weighed nearly 10·3 kilograms (twenty-three pounds). In addition, the child had become bright and cheerful, speech had improved, and once again he showed an inclination to walk. The abdomen was slightly protuberant, but the stools were now fairly normal.

DR. KINGSLEY NORRIS thought that some abdominal pathology was the underlying condition, with subsequent infantilism, and he suggested that megacolon be excluded. He thought that the thyreoid extract had temporarily stimulated the child's metabolism; possibly relapse would occur.

Scurvy.

DR. LIONEL HOOD showed a male child, aged eight months, with a seven weeks' history of fever, irritability, fretfulness, and screaming if his limbs were touched. He had been born at full term, and had been breast-fed for two weeks, then on boiled skimmed cow's milk up to the time of his admission. He had been given orange juice—a teaspoonful twice a day—until six weeks prior to admission, when he had refused it. Examination revealed a pale baby, who screamed if his limbs were handled. All the long bones were tender, especially in the lower extremity. There was a swelling of both wrist joints and some apparent beading of the ribs. There were some submucous hæmorrhages at the bases of the upper incisor teeth and in the palate. No other abnormality was found.

The wrist joints were examined by X rays and the report was that the appearances were consistent with healed rickets. Because of this report and the apparent rosary, Dr. Hood said that he had intended to show the patient as a sufferer from scurvy and rickets, but subsequent X ray examination of the lower limbs showed the typical radiological appearances of scurvy; the apparent beading of the ribs was due to a depression of the sternum. The explanation was to be found in excessive boiling of the milk with inadequate vitamin replacement. When given orange juice in a dose of fourteen cubic centimetres (four fluid drachms) four times a day, the child made an uneventful recovery.

DR. COLIN MACDONALD discussed the radiological appearances of scurvy as shown in the lower end of the femur in Dr. Hood's case. There were the Wimberger ring round the epiphysis, the ground-glass appearance of the shaft, with thinning of the cortex, the increase in the zone of temporary calcification known as the Trummerfeld zone, and the subperiosteal and epiphyseal hæmorrhages. This case showed that it was safer to base an opinion on the radiological appearances of the lower limb rather than the upper, as the changes were usually more pronounced in the lower. He now agreed that Dr. Hood's case was one of uncomplicated scurvy.

Splenomegaly.

DR. R. N. HOWARD showed a girl, aged seven years, who had suffered from anæmia for the previous seven months and pain in the left hypochondrium for one week. At the time of examination the child was very anæmic and the conjunctivæ slightly icteric. The liver was enlarged for three fingers' breadths below the costal margin, and the spleen extended to the level of the anterior superior iliac spine. It was firm and not tender. Her temperature was 39·4° C. (103° F.) and subsided by lysis in a week. The cause of this was not determined. The red blood cells numbered 2,500,000 per cubic millimetre; the hæmoglobin percentage was 65 and the colour index 1·3. The appearances of the blood were typical of pernicious anæmia. The platelet count was 130,000 per cubic millimetre. The coagulation and bleeding times were normal. There was marked hæmolysis of the red cells in 0·625% saline solution. The urine contained no bile salts, but a slight excess of urobilin. Fouchet's test was faintly positive.

The blood serum contained 3·2 units of bilirubin to Van den Bergh's test. There was no free hydrochloric acid in the stomach.

Half a pound of cooked liver was given daily, and there was great and rapid improvement, so that in two weeks' time the numbers of red cells rose to 4,000,000 per cubic millimetre, the hæmoglobin content was 75%, and the colour index was 0·9. Macrocytosis was still evident, but no other abnormality. As the child was in such an improved condition, splenectomy was performed, and a month later the red cell count was 4,500,000 per cubic millimetre, the hæmoglobin percentage 80, and the colour index 0·9.

Dr. Howard said that the points of interest about this child were the initial pyrexia which had never been explained, and the acute blood crisis, which was not hæmolytic, because the response to pigment tests had been normal. It was not aplastic, because there were signs of regeneration. The blood film had been typical of pernicious anæmia, the colour index had been 1·3, and there had been no free hydrochloric acid in the stomach, and the child had rapidly improved on liver therapy. This was apparently a case of acquired hæmolytic splenomegaly.

Dr. Howard also showed a female child, aged nine years, symptomless herself, but admitted to hospital because the mother had splenomegaly and jaundice, and one brother and two sisters had hæmolytic splenomegaly. Examination revealed a firm, palpable spleen, a red blood cell count of 5,200,000 per cubic millimetre, and a hæmoglobin percentage of 80. The red cells commenced to hæmolyse in a 0·625% solution of saline, and completely hæmolysed in a 0·45% solution. Two months later splenectomy was performed, and at the time of the meeting, five months later, the red blood cell count was 4,200,000 per cubic millimetre, the hæmoglobin percentage was 80, and the colour index 1·0. The red blood cells did not then commence to hæmolyse until placed in a 0·55% solution of saline.

Dr. Howard remarked that there had been a decrease in the fragility of the red cells following splenectomy, a point about which there was considerable debate at the present time.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

AT a regular monthly meeting of the Senate of the University of Sydney, held on Monday, May 2, 1932, the following degrees were conferred *in absentia*:

Bachelor of Medicine and Bachelor of Surgery: John Glasgow Morris, John Horton Young.

The Diploma in Public Health was awarded to James Inglis Robertson, M.B., B.S.

Permission was granted for the holding of examinations for the Diplomas in Public Health and Tropical Medicine during the week commencing May 30, 1932.

Dr. E. W. Fairfax was appointed as the delegate of the University to attend the centenary celebrations of the British Medical Association to be held in London in July next.

The resignation of Dr. H. R. G. Poate, Lecturer in Clinical Medicine at the Royal Prince Alfred Hospital, was received and accepted with regret. The Senate decided to record its appreciation of the services rendered by Dr. Poate during his tenure of the lectureship.

The following appointments were approved: Dr. J. H. Cramsie, Dr. D. W. Magill and Dr. S. G. Nelson as Honorary Demonstrators in Anatomy. Dr. Cecil Rowntree, of the Cancer Hospital, London, as Deputy Representative of the Cancer Research Committee on the Grand Council of the British Empire Cancer Campaign. Dr. R. J. Taylor (as Honorary Assistant Physician), Dr. L. F. Dods (as Honorary Relieving Assistant Physician), and Dr. G. Norrie (as Honorary Dermatologist) to the Honorary Medical Staff of the Royal Alexandra Hospital for Children. Dr. Alan Oxenham and Dr. E. W. Frecker as Honorary Assistant Radiologists to the Honorary Medical Staff of Saint Vincent's Hospital.

It was resolved to admit Mr. J. S. Baird, B.D.S., to the degree of Doctor of Dental Science (D.D.Sc.) for his thesis entitled: "Infective Complications of Fracture of the Mandible", which the examiners considered was an original contribution of distinguished merit, adding to the knowledge of oral surgery. (Mr. Baird took the degree of Bachelor of Dental Surgery in 1927 with second class honours.)

The By-Laws of the Faculty of Dentistry were, on the recommendation of the Faculty of Dentistry, amended to provide for the examination of first year students in anatomy in addition to chemistry, physics and metallurgy.

Naval and Military.

PRESENTATION OF THE AUSTRALIAN ARMY MEDICAL CORPS KING'S COLOURS.

AN interesting ceremony was carried out at the Medical Society Hall, Albert Street, East Melbourne, on December 16, 1931, when the colours presented to the Australian Army Medical Corps by His Majesty King Edward VII were taken under the temporary care of the Victorian Branch of the British Medical Association.

The ceremony of handing over the colours was performed by the Director General of Medical Services, Major-General George W. Barber, C.B., C.M.G., D.S.O., V.D. All officers of the Australian Army Medical Corps were invited to attend, and a large gathering of officers and their wives and friends assembled, together with the members of the Council of the Victorian Branch of the British Medical Association.

Representatives were present from the following units and services: All Field Ambulances, the Australian Army Medical Corps, Defended Ports, Field Hygiene Sections, Regimental Medical Officers, Dental Services, Army Nursing Service, Pharmaceutical Service, Quartermasters, and the Australian Instructional Corps attached to the Australian Army Medical Corps.

The colour escort consisted of members of the Royal Australian Army Medical Corps, in charge of Major Tait, Staff Officer to the Deputy Director of Medical Services. The colour-bearer was the junior Lieutenant-Colonel on the active list, Lieutenant-Colonel A. P. Derham, M.C.

The entry of the colour party was preceded by a fanfare of trumpets. As the colour party advanced to the dais, the Director General of Medical Services instructed the Deputy Director of Medical Services, Colonel R. M. Downes, C.M.G., V.D., to hand the colours to the President of the Victorian Branch of the British Medical Association, Lieutenant-Colonel B. M. Sutherland, O.B.E., A.A.M.C.

Colonel Downes prefaced the presentation with the following address:

> The Director General desires me to make an explanation of the circumstances attending this ceremony. The King's Colours before you were presented by His Majesty King Edward VII to the Australian Army Medical Corps in recognition of its services during the South African Campaign of 1899. The consecration and presentation were carried out at a Royal review held in Albert Park on November 14, 1904, by His Excellency the Governor-General, Lord Northcote. At the same time King's Colours were presented to eighteen Light Horse Regiments and the Royal Australian Artillery. These colours were received by three representatives of the Australian Army Medical Corps from New South Wales, the State which had had the greatest share in the winning of this signal honour. We are honoured to have here tonight Colonel A. H. Sturdee, who was such a distinguished member of the Australian Army Medical Corps in the South African War.
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> It may not be out of place here to make a few remarks on the significance of colours. Their use dates back to remote antiquity, evidence having been found of their existence in India over 5,000 years ago. We are all familiar too with the vexilla of the Roman

legions, fashioned in metal, and it is of interest to note that just prior to the Christian era the material was altered to cloth, silk or damask. Like tribal and national flags, colours serve as symbols embodying the spirit of the people who fight under them. In the past two purposes have been served by them. In the first place to indicate rallying points on the field of battle, and many epic stories of heroism are attached to their defence. The frequently used expression, "keep the flag flying", is derived from this. With changes in the weapons of war colours have changed from their essential nature in relation to hand-to-hand combat to that of symbols of sentimental and patriotic feeling. Their last appearance in a European war was in the Crimea, where the mortality of the colour-bearers was tremendous. Their last British appearance on the battlefield was at Isandlwana in the Zulu War of 1879. Both armies carried them in the Russo-Japanese War. Since then they have not been carried into battle.

They have also been used as distinguishing marks of persons of especial importance in social or military spheres. From being unlimited and uncontrolled in numbers and variety, there are now in the British Army only three varieties of colours, the guidons of cavalry regiments and the King's and regimental colours of infantry battalions.

The regimental colours are inscribed with the battle honours of the unit, the King's Colours are the great Union Jack which we have here, but nowadays bear in the centre an imperial crown with the name of the unit.

As far as can be ascertained, the grant of King's Colours to the Australian Army Medical Corps is the only known instance in which a medical service has been so signally honoured.

The colours which are being handed over tonight remained in New South Wales until 1929, when they were brought to Army Headquarters with a view to their future custody in the cathedral at Canberra.

It is now the intention of Army Headquarters to entrust them temporarily to your care. They bear the inscription: "Presented by His Most Gracious Majesty the King Emperor, to the Australian Army Medical Corps in recognition of services rendered to the Empire in South Africa, 1904."

I have now, Sir, formally to request you to accept the honour of the custody of the King's Colours presented to the Australian Army Medical Corps.

Taking the colours from the colour-bearer, Colonel Downes handed them to the President of the Victorian Branch of the British Medical Association, who formally accepted them for safe keeping under the temporary care of the Branch. The colour party presented arms and the trumpeters sounded "The Royal Salute".

The President then proceeded at the head of the colour party and placed the colours in position in the foyer of the hall.

Correspondence.

DANGEROUS CONFUSION.

SIR: The medical profession is vitally interested in the coining of new names for pharmaceutical products. The dangerous confusion which is occurring may lead to the loss of life through the innocent action of a chemist. To the public and pharmacists the matter is, therefore, of much moment. The comparison of the following list of duplicated trade names (not necessarily trade marks) makes our point clearer.

"Hydriol": a solution of hydriodic acid, also an iodized oil.

"Felsol": a cresylic acid solution, also an asthma powder for oral use.

"Tenax": a carbolized tow, also an antiseptic soap.

And this list could be multiplied many times. But an equally dangerous risk arises through the similarity of coined names for different articles, not merely different makers. The risk will appear to be much greater when these trade words are written hurriedly.

"Percain"—"Procain", "Alocol"—"Oleocal", "Hormonal"—"Hormonol", "Ammonol"—"Allonal", "Parawax"—"Porowax", "Lipiodol"—"Lipoiodine".

We suggest that some official action should be taken to avoid these risks, which should not be incidental to the harmony existing at present between your readers and pharmacists, to the possible danger of the unsuspecting patient.

Yours, etc.,

HENRY FRANCIS AND COMPANY.

Melbourne,
May 3, 1932.

OBSTETRICAL RADIOGRAPHY.

SIR: In your issue of May 14, 1932, Dr. Flecker expresses "amazement and astonishment" at the letter of Dr. Davidson of a previous issue. Despite Dr. Flecker's assurance that injury to the fœtus is a bogy, I would ask his pardon to allow me to be quite unconvinced.

I fully realize the difficulties of the path that leads to scientific truth, but I believe that one who follows carefully the teachings of Professor Windeyer will arrive in 99% of cases at just as accurate a knowledge as one who uses radiography in obstetrical diagnosis. He will have too, or should have, the gratifying feeling that he has not put his patient to what is, in my opinion, an unjustifiable and unnecessary expense.

Yours, etc.,

H. O. LETHBRIDGE.

Narrandera,
May 13, 1932.

SIR: I regret that Dr. Flecker should consider any criticism I have offered unfair.

To call a reasonable fear of damage to such delicate tissue as that of the growing fœtus, "a bogy" does not help and is certainly an unscientific attitude to adopt.

Dr. Flecker in his article in THE MEDICAL JOURNAL OF AUSTRALIA (October 6, 1926) writes: "Numerous reports are given in the literature of effective treatment . . . when the pregnancy is terminated in the birth of a normal and healthy child, both physically and mentally."

In my opinion it is unscientific to claim that any medical man can tell whether a new-born child is normal mentally; even at four years of age it is extremely difficult.

My view of the subject is that obstetrical radiography should only be used in very carefully selected cases, as it cannot be proved in the present state of our knowledge that radiation of the embryo or fœtus is entirely free from the possibility of damage to the growing tissue. Further, that every case of radiation during pregnancy should be carefully followed up as to the ultimate development of the child, even to adult life when possible.

Yours, etc.,

A. M. DAVIDSON.

Enmore,
New South Wales,
May 15, 1932.

Obituary.

RICHARD ARTHUR.

WE regret to announce the death of Dr. Richard Arthur, which occurred at Mosman, New South Wales, on May 21, 1932.

NOTICE.

IN a notice recently sent to members of the New South Wales Branch of the British Medical Association, the time of the Clinical Meeting to be held at the Royal North Shore Hospital, St. Leonards, on June 9, 1932, was wrongly stated as 8 p.m. Members are requested to note that the meeting will be held at 3 p.m.

A meeting will be held in the lecture theatre of the Royal Alexandra Hospital for Children, Camperdown, on Monday, May 30, 1932, at which papers will be read by Dr. R. B. Wade, Dr. E. H. M. Stephen, Dr. F. Tidswell, Dr. Karen Helms and Dr. J. Steigrad, on the acute anterior poliomyelitis epidemic of 1931-1932. The honorary medical staff of the hospital extends an invitation to all medical practitioners to attend this meeting.

Books Received.

GENERAL PRACTICE SERIES: CHEST DISEASES IN GENERAL PRACTICE, WITH SPECIAL REFERENCE TO PULMONARY TUBERCULOSIS, by P. Ellman, M.D., M.R.C.P., with Foreword by S. L. Cummins, C.B., C.M.G., M.D.; 1932. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 281, with 132 illustrations. Price: 15s. net.

Diary for the Month.

JUNE 1.—Western Australian Branch, B.M.A.: Council.
JUNE 1.—Victorian Branch, B.M.A.: Council.
JUNE 2.—South Australian Branch, B.M.A.: Council.
JUNE 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 9.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 10.—Queensland Branch, B.M.A.: Council.
JUNE 14.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 15.—Western Australian Branch, B.M.A.: Branch.
JUNE 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 22.—Victorian Branch, B.M.A.: Council.
JUNE 24.—Queensland Branch, B.M.A.: Council.
JUNE 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. R. M. Murray has been appointed Acting Government Medical Officer at Townsville and a Health Officer for the purposes of *The Health Acts*, 1900 *to* 1931, and Medical Officer to the State Children Department, Townsville, Queensland.

* * *

Dr. S. E. Holder (B.M.A.) has been appointed an Honorary Medical Officer to the Wallaroo Hospital, South Australia.

* * *

Dr. D. Zacharin (B.M.A.) has been appointed Certifying Medical Practitioner at East St. Kilda, Victoria, pursuant to the provisions of the *Workers' Compensation Act*, 1928.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xiv.

LAUNCESTON PUBLIC HOSPITAL, TASMANIA: Resident Medical Officer (male).

MATER MISERICORDIÆ HOSPITAL, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

RACHEL FORSTER HOSPITAL FOR WOMEN, SYDNEY, NEW SOUTH WALES: Honorary Psychiatrist, Honorary Temporary Relieving Assistant Surgeon.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Assistant Radiologist, Honorary Radiologist.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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